# "SEROPREVALENCE OF HEPATITIS B, HEPATITIS C & SYPHILIS AND LIVER ENZYMES LEVELS IN HIV POSITIVE PATIENTS ATTENDING STD CLINIC"

**Dissertation submitted in**

**Fulfillment of the University regulations for**

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**(BRANCH XX)**


**MADRAS MEDICAL COLLEGE**

**THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY**

**CHENNAI**

**APRIL 2016**

# CERTIFICATE

Certified that this dissertation titled ***"SEROPREVALENCE OF HEPATITIS B, HEPATITIS C & SYPHILIS AND LIVER ENZYMES LEVELS IN HIV POSITIVE PATIENTS ATTENDING STD CLINIC"*** is a bonafide work done by **SUNITHA.N,** Postgraduate student of the Department of Dermatology, Venereology and Leprosy, Madras Medical College, Chennai – 3 during the academic year 2013 – 2016. This work has not previously formed the basis for award of any degree.

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# DECLARATION

I **Dr. SUNITHA.N** solemnly declare that the dissertation on ***"SEROPREVALENCE OF HEPATITIS B, HEPATITIS C & SYPHILIS AND LIVER ENZYMES LEVELS IN HIV POSITIVE PATIENTS ATTENDING STD CLINIC"*** was done by me at Madras Medical College during 2013-2016 under the guidance and supervision of **Prof**. **Dr.S. KALAIVANI, M.D.,DV,** Incharge Director and Associate Professor , Institute of Venereology, Madras Medical College/RGGGH, Chennai- 600003.

The dissertation is submitted to the Tamil Nadu DR.MGR Medical University towards the partial fulfillment of the rules and regulations for the award of **M.D Degree in Dermatology, Venereology and Leprosy (BRANCH – XX).**

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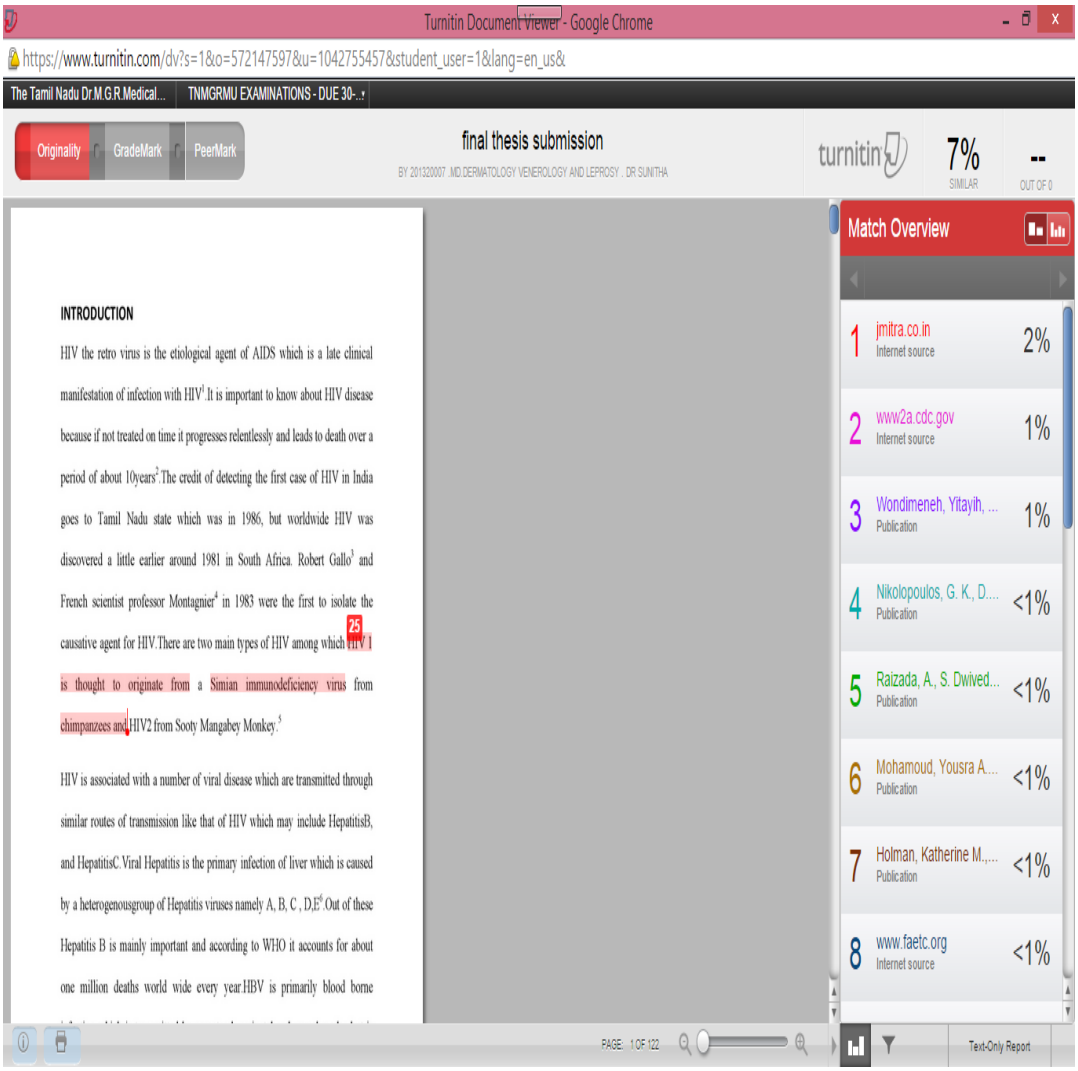
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INTRODUCTION
HIV the retro virus is the etiological agent of AIDS which is a late clinical manifestation of infection with HIV[1].It is important to know about HIV disease because if not treated on time it progresses relentlessly and leads to death over a period of about 10years[2].The credit of detecting the first case of HIV in India goes to Tamil Nadu state which was in 1986, but worldwide HIV was discovered a little earlier around 1981 in South Africa. Robert Gallo[3] and French scientist professor Montagnier[4] in 1983 were the first to isolate the causative agent for HIV.There are two main types of HIV among which HIV 1 is thought to originate from a Simian immunodeficiency virus from chimpanzees and HIV2 from Sooty Mangabey Monkey.[5]
HIV is associated with a number of viral disease which are transmitted through similar routes of transmission like that of HIV which may include HepatitisB, and HepatitisC.Viral Hepatitis is the primary infection of liver which is caused by a heterogenousgroup of Hepatitis viruses namely A, B, C , D,E[6].Out of these Hepatitis B is mainly important and according to WHO it accounts for about one million deaths world wide every year.HBV is primarily blood borne
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# CONTENTS

# SEROPREVALENCE OF HEPATITIS B, HEPATITIS C & SYPHILIS AND LIVER ENZYMES LEVELS IN HIV POSITIVE PATIENTS ATTENDING STD CLINIC


## ABSTRACT

### OBJECTIVES AND AIMS OF THE STUDY:

To detect the Prevalence of Hepatitis B, Hepatitis C and Syphilis and detection of Liver enzymes levels in HIV positive patients and thereby to establish the link of Liver enzyme levels by LFT in Hepatitis infection and HIV positive patients and also in patients who are VDRL reactive and HIV positive.

### MATERIALS AND METHODS:

100 cases of HIV positive patients attending the STI Out Patient detected by Rapid Assay(immunoblot assay)kit would be selected for this study.

Detailed clinical history (including H/O of presenting complaints, occupational history, menstrual history, marital history, sexual history/ last H/o contact, obstetric history, past H/o sexually transmittedinfections) followed by thorough clinical evaluation would be done. 5 ml blood is withdrawn aseptically from the patient. The serum is separated and subjected to Rapid Assay test for the detection of HIV and then HIV positive blood samples are subjected to VDRL, HBsAg, anti-HCV,.and LFT.VDRL reactives will be subjected to TPHA test. 10 patients who are HIV negative are taken as controls

### RESULTS:

In our study, HIV-HBV co-infection was seen in 13(13%) patients, HIV-HCV co-infection was seen in 1(1%) patient, HIV with Syphilis co-infection was seen in

2(2%) patients, Elevated liver enzyme levels in HIV positive patients were seen in 4(4%) patients, Decreased CD4 count was seen seen in 3(3%) patients, HIV-HBV co-infection with elevated liver enzyme levels and decreased CD4 count was seen in 3(3%) patients, HIV-HCV co-infection with elevated liver enzyme levels and decreased CD4 count was seen in 1(1%) patient, HIV-HBV-HCV co-infection with elevated liver enzyme levels and decreased CD4 count was seen in 1(1%) patient

**CONCLUSION**

Thus to conclude, The decreased CD4 count with elevated liver enzyme levels as seen in our study will cause further liver damage, hence ART should be immediately started in these patients. Hence routine screening for Hepatitis B and Hepatitis C should be emphasized in HIV positive patients since its early detection can decrease the morbidity and mortality due to liver damage among these patients and screening for Syphilis should also be encouraged in HIV positive patients since its early detection can decrease the morbidity and mortality among these patients. By routine screening effective treatment can be implemented to increase the life span and quality of life among such patients .

**KEYWORDS:**

Human Immunodeficiency Virus, Hepatitis B, Hepatitis C , Syphilis, elevated Liver enzymes, CD4 count.

# INTRODUCTION

HIV the retro virus is the etiological agent of AIDS which is a late clinical manifestation of infection with HIV[1].It is important to know about HIV disease because if not treated on time it progresses relentlessly and leads to death over a period of about 10years[2].The credit of detecting the first case of HIV in India goes to Tamil Nadu state which was in 1986, but worldwide HIV was discovered a little earlier around 1981 in South Africa. Robert Gallo[3] and French scientist Professor Montagnier[4] in 1983 were the first to isolate the causative agent for HIV. There are two main types of HIV among which HIV 1 is thought to originate from a Simian immunodeficiency virus from chimpanzees and HIV2 from Sooty Mangabey Monkey.[5]

HIV is associated with a number of viral disease which are transmitted through similar routes of transmission like that of HIV which may include Hepatitis B, and Hepatitis C. Viral Hepatitis is the primary infection of liver which is caused by a heterogenous group of Hepatitis viruses namely A, B, C , D,E[6].Out of these Hepatitis B is mainly important and according to WHO it accounts for about one million deaths worldwide every year.HBV is primarily blood borne infection which is transmitted by parenteral, perinatal and sexual modes but is also excreted

in saliva, breast milk, urine, bile, faeces, semen and vaginal secretions, among these saliva and semen are well known to transmit HBV infection[7].Hepatitis C is epidemiologically similar to Hepatitis B and is known to occur only in humans. It is mainly transmitted by blood transfusion, injection drug abusers, immunocompromised and transplant recipients are at major risk and accounts for one quarter of chronic Hepatitis in India.[9] Thus although Hepatitis B and Hepatitis C are mainly transmitted through blood transfusion and blood products, sexual transmission is also known to occur though this mode of infection is low and have become epidemiologically important from the heterosexual transmission point of HIV.

Syphilis is an infectious disease caused by Treponema Pallidum which belongs to the order Spirochaetals and family Spirochaetacea which has a chronic course and capacity to affect any structure of the body, which may show florid manifestation or might remain asymptomatic for many years and it also resembles many disease in medicine or surgery fields, apart from transmission to offspring its transmission to laboratory animals can also occur which can be treated to the extent of presumptive cure[14].

Syphilis is complexly related to HIV infection, there are many documental studies stating that Sexually Transmitted Infection's (STIs)

like Syphilis are thought to raise the risk of HIV infection among the individuals of homosexuals and also heterosexuals.[15,16] Hepatobiliary system is somehow uniquely related to retroviral infection and viral hepatitis and Syphilis. Liver enzymes are variably elevated in HIV infected individuals which was more prevalent prior to the invent of HAART(Highly Active Antiretro Viral Therapy),but now apart from ART, increase in liver enzyme is associated to the underlying primary liver pathology, alcohol and also to viral Hepatitis.[19]Thus abnormal liver enzyme in HIV disease can present with a wide range of manifestation from mild steatohepatitis to the extremes of liver fibrosis hence its early detection helps to asses the prognosis in such retroviral infection,[20]but it should also be beared in mind that it is possible for HIV patients to show elevated liver enzymes even in the absence of Hepatitis.[21]

Unlike HIV, liver enzymes are not significantly elevated in Syphilis, but there are literature stating the occurence of Syphilitic hepatitis in HIV infected patients.[22-25]Early sign to detect the co-exsistance of syphilitic hepatitis are, the patient presents with perianal lesion and disproportionate elevation in liver enzyme levels.[26]

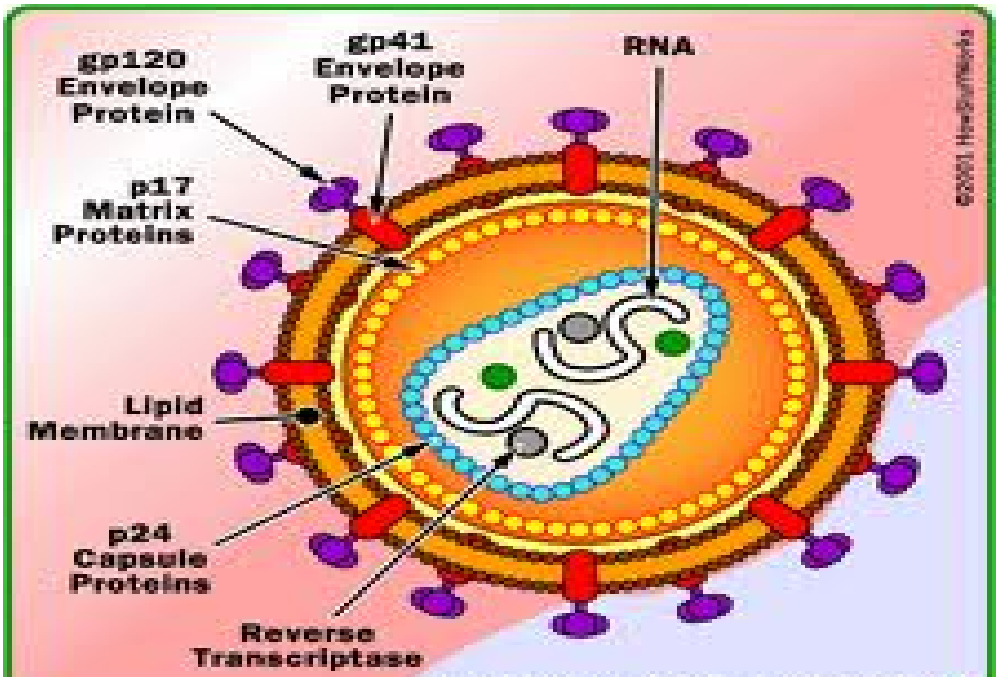
# *Review of Literature*

# REVIEW OF LITERATURE

## STRUCTURE OF HUMAN IMMUNODEFICENCY VIRUS (HIV)

Hockley[27] and Gelderblomet at [28] have contributed for their discovery of HIV structure.It is a spherical shaped retrovirus which encloses a nucleocapsid which is roughly bullet shaped which contains three different enzymes reverse transcriptase,integrase and protease and a single standard RNA. A lipid envelope surrounds the viral core,these envelope bears raised spike like projection called gp160,which inturn consists of cap like projection called gp120 .

**Figure 1: HIV structure**



**REPLICATION CYCLE OF HIV**

HIV is a retrovirus which is unique in a way that it replicates only in human cells .

The principle steps in the beginning of HIV replication are

1. The viral entry into target cell which occurs by facilitating the binding of gp120(envelope glycoprotein) of the virus to a unique receptor(CD4)

↓

2. A process of conformational change exposes gp41(fusion peptide) which favours the intimate fusion of virus with cell surface membrane

↓

3. This fusion is followed by reverse transcription which occurs in the viral nucleoprotein, where it generates a single DNA copy from the viral genome , this step is accomplished by the help of enzymes, reverse transcriptase

↓

4. Integrase enzyme is the one that facilitates the complex integration of viral particle into the chromosomal DNA of host cell which forms the ‘provirus’ .

↓

5. viral protein expression takes place by the collaborative action of RNA polymerase, transcription factor and also by certain regulatory proteins(tat, rev).

↓

6. Assembly of core enzymes of HIV and RNA takes place but they are still immature due to the lack of envelope protein which are synthesized in the specialized organelles of endoplasmic reticulum of the infected cells, by this way viral envelope protein is expressed on to the surface of cell

↓

7. The process of replication ends when the enzyme protease cleaves certain proteins like gag, gag-pol so that the virion matures and the matured virion are now ready to restart a new cycle.

**ROUTES OF TRANSMISSION**

Venereal (semen, vaginal and cervical discharge ) and blood tansfusion,[29] are the two main modes of HIV transmission, but the virus can also make entry into the body through following ways :

- Breast milk[30]
- Saliva[30]
- Colostrum[30]
- Urine[30]
- Tears[30]
- Presence of genital ulcer extends the risk of retroviral transmission[31]

- HIV transmission are also known to occur through artificial insemination[32]
- Vertical transmission becomes greater during 3rd trimester or at the time of delivery[33]
- Anticipation of infected mother transmitting the infection to new born is about 20% to 50%[34,35]
- Blood donation from high risk group are not reliable since the patient might be in window period(3-17weeks) or the result can also be false negative.[36,37]
- The possibility of transmission through needle stick injury in health care workers is about 0.5%[38]

**PATHOGENESIS**

The retro virus enters the human body via the mucosal surface like the, genital mucosa, oropharynx, rectum primarily by sexual contact and since these mucosal surface are rich in langerhans cells and dendritic cells they trap the antigen and virus particles which replicates rapidly and subsequently establishes primary viraemia by then a huge number of infected cells in peripheral blood and high titres of virus of upto several million virus particles per ml of blood can be detected.[39]

This response is followed by significantly huge fall in the number of CD4 cells, and an associated activation of $CD8^+$ T cells, which is

known to kill virus infected cells, and subsequently produce seroconversion. This $CD8^+$ T cell plays an important role in controlling virus level. This is followed by a clincal latent period which may last for several years. The exact mechanism how HIV destroys CD4+ T cells is quite unexplained and complex[40]

The possible known mechanism are:

**Direct cell killing**

When large number of viruses are produced and burst out from cell surface theymay directly kill infected CD4+ Tcells.

**Syncytia formation**

The infected CD4+ T cells are known to fuse with neighbouring uninfected cells to form ballon like large cells called syncytia.

**Apoptosis**

The infected CD4+ cells may also be killed by programmed cell death called apoptosis which can occur either in blood stream or lymph node.

Eventhough new T cells are persistently being produced by the thymus for the replacement of the lost old ones, the regenerative ability of the thymus is being slowly destroyed by direct infection of thymocytes by the retro virus, ultimately even the low number of $CD4^+$ T

cells required to maintain the proper immune system is lost, leading to full blown AIDS.

## CLINCAL MANIFESTATION

Clinical features of HIV can be grouped into the following stages

1. Primary HIV infection
2. Asymptomatic stage
3. Early symptomatic stage
4. Latesymptomatic stage
5. Advanced HIV disease.

### 1. Primary HIV infection

It usually occurs in 50 to 90% patients around 2 to 4weeks after the viral exposure and may present with features resembling infectious mononucleosis like pharyngitis, fever, lymphadenopathy, maculopapular rash, head ache nausea, vomiting, myalgia[42],oral ulcer, arthalgia[42], diarrhoea, neurological features – radiculopathy, meningoencephalitis, peripheral neuropathy, and psychosis.

### 2.Asymptomatic stage

Primary HIV infection is followed by an asymptomatic stage during which the patient is symptomless except some patient can present with persistent generalised lymphadenopathy.

**3. Early symptomatic stage**

**a) Constitutional symptoms** are present initially like fatigue, fever, unexplained weight loss, recurrent diarrhoea.

**b) Cutaneous features:**

(i) Infectious manifestation:

- Oral candidiasis
- Molluscum contagiosum[41]
- Oral hairyleukoplakia
- Wart
- Dermatophytosis[43]
- Herpes zoster[43]
- Herpes simplex virus infection[43]

(ii) Non infectious manifestation:

- Psoriasis vulgaris[46]
- Reiters syndrome
- Seborrhoeic dermatitis
- Pruritic papular dermatitis
- Vitilligo[48]
- Pitryriasis rubra pilaris[49]
- Photodermatitis[47]

- Granuloma annulare[48]
- Nail changes-leuconychia, onychomycosis, paronychia

**c) Respiratory features include -** Pulmonary tuberculosis, haemophilus influenza, streptococcus pneumonia and mycoplasma pneumonia

**d) Haematological features-**anemia[45], thrombocytopenia , neutropenia

**e) Renal manifestation** - glomerulonephritis, HIV associated nephropathy and nephrotic syndrome.

**4. Late symptomatic stage**

A drop in the CD4 count triggers opportunistic infections like pneumocystis carinii pneumonia(PCP), mycobacterium avium complex, toxoplasmosis, oesophageal candidiasis.

**5. Advanced HIV disease**

This includes malignancies like kaposi's sarcoma, lymphoma, Non Hodgkins Lymphoma(NHL)[44], among these kaposi's sarcoma was supposed to be the first and most common neoplasm occurring in HIV disease. Other opportunistic infection reported to occur in advanced HIV disease are CMV infection, cryptococcal meningitis, histoplasmosis and AIDS wasting syndrome.

**Laboratory diagnosis of HIV**

1.Detection of HIV specific antibodies in plasma/serum by the following test

**A) Screening test which primarily include**

- ELISA

ELISA are of four main type

i. Indirect ELISA
ii. Sandwich ELISA
iii. Competitive ELISA
iv. Antigen-Antibody capture ELISA

- Rapid test for HIV

**B) Supplemental tests which include**

- Western blot test
- Immunofluorescence assay
- Line immune assay

2. Detection of HIV specific antibodies in body fluids like saliva

3. Detection of HIV specific antibodies in patients urine

4. Confirmatory test

a. Polymerase Chain Reaction(PCR)

b. Virus isolation

c. Test for detection core antigen (P24)

**Figure 2: ELISA plate**

**Figure 3:INDIRECT ELISA**

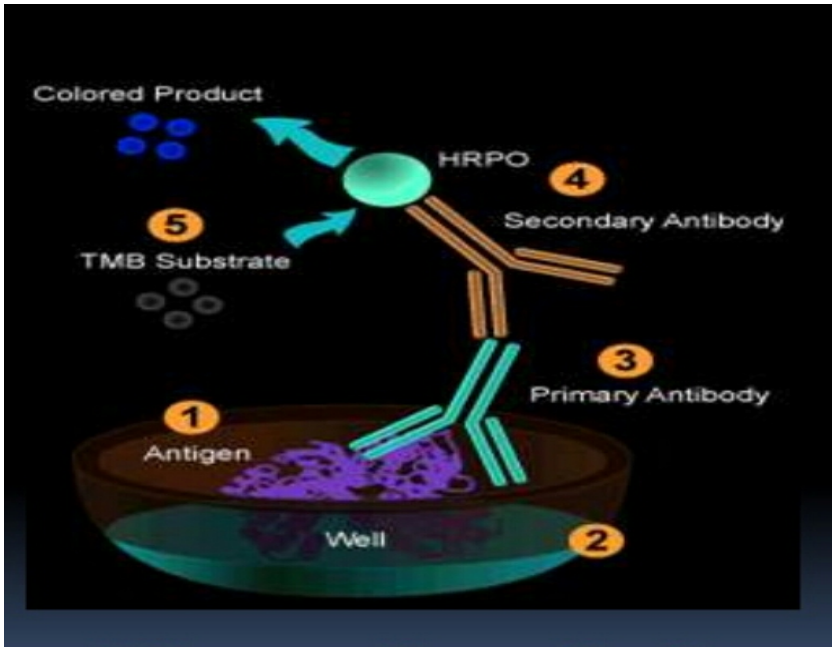
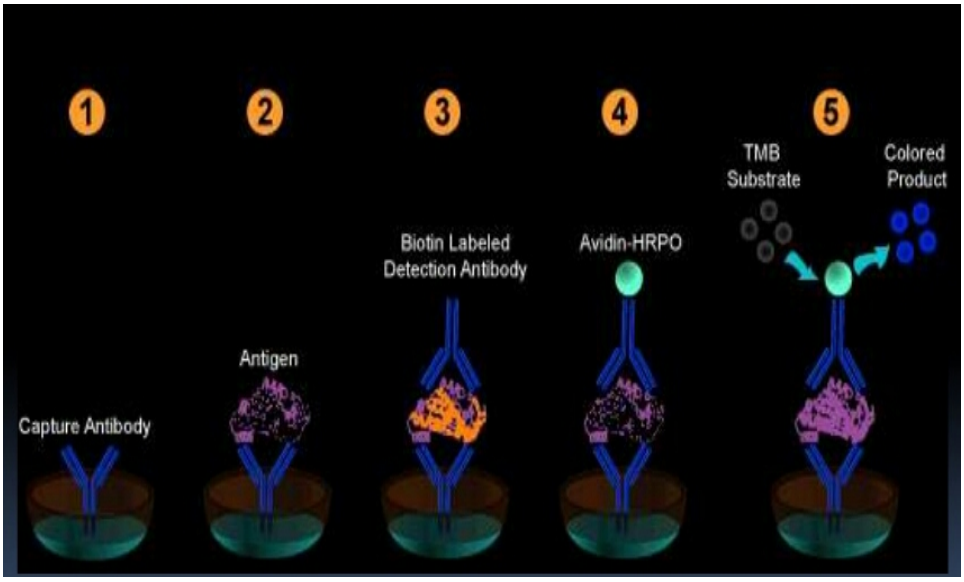
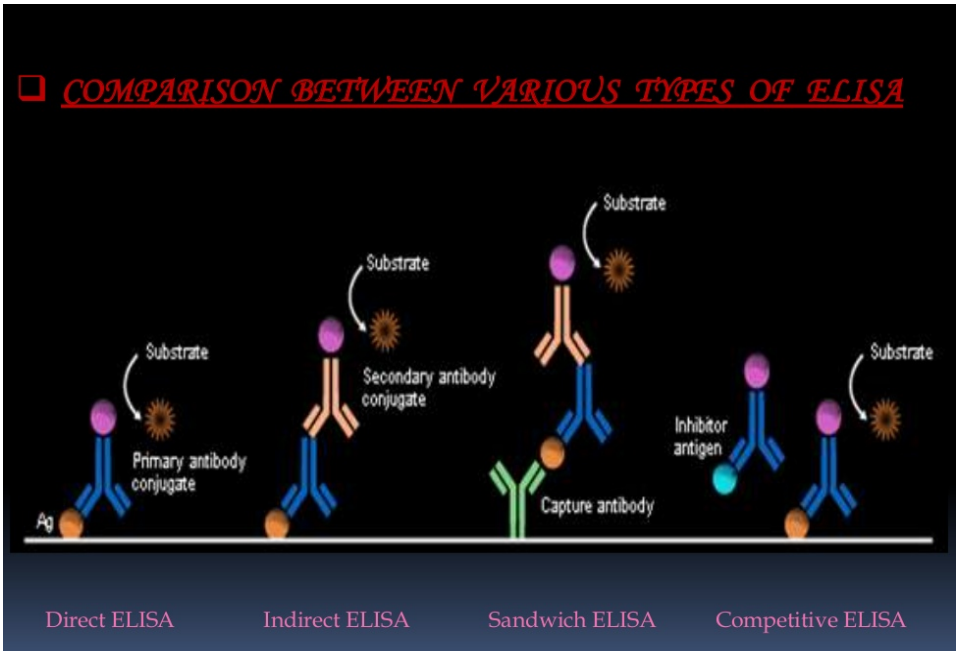


**Figure 4:SANDWITCH ELISA**



**WHO guidelines for starting ART based on clinical staging and CD4 count**

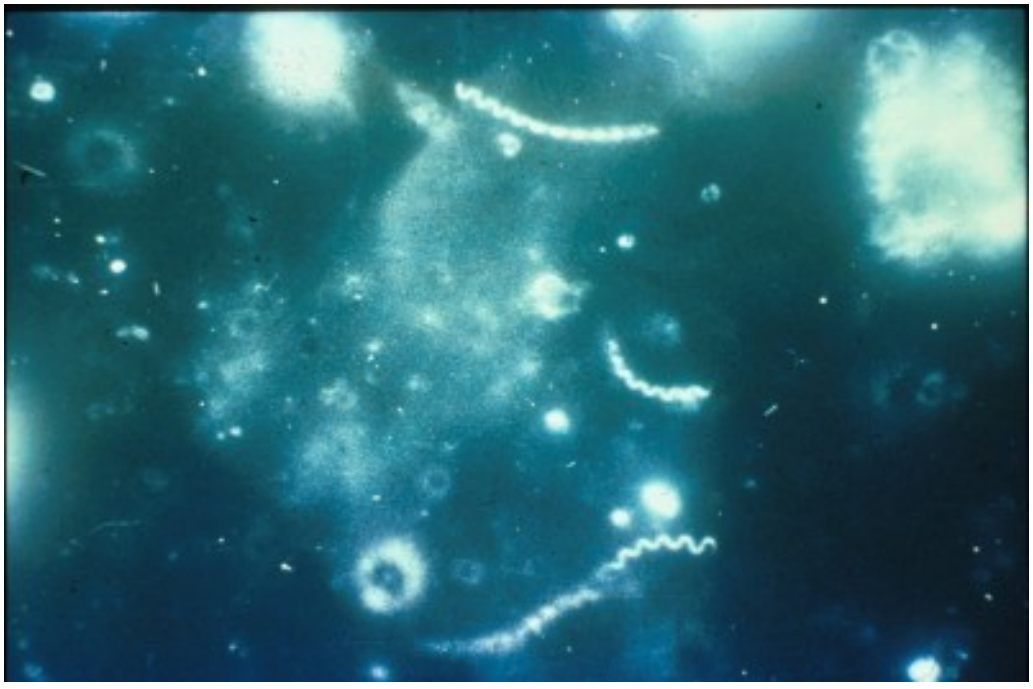
| Clinical staging | Recommendation |
|---|---|
| Clinical stage I and II | ART started if CD4 count is<350cells/mm3 |
| Clinical stage III and IV | ART started irrespective of CD4 count |
| **For HIV patients co-infected with Hepatitis B and Hepatitis C infection** | |
| HIV-HBV,HIV-HCV co-infection in the absence of chronic active Hepatitis | ART started if CD4 count is<350cells/mm3 |
| HIV-HBV,HIV-HCV co-infection in the presence of chronic active Hepatitis | ART started irrespective of CD4 count |
| **For HIV patients co-infected with Tuberculosis** | |
| HIV-TB (pulmonary or extrapulmonary) co-infected patients. | ATT must be started immediately and ART can be added between 2 weeks to 2 months or as early as possible when TB treatment is tolerated |

**Syphilis**

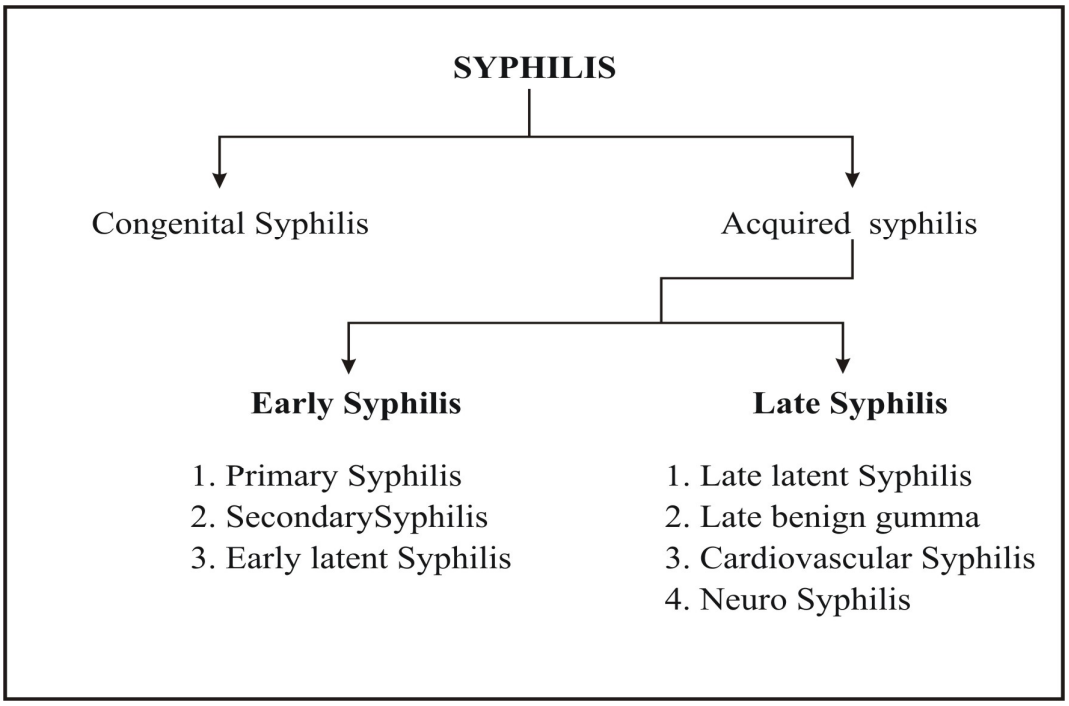
**Morphology**

Treponema Pallidum the causative agent of Syphilis is a regular spiral shaped, coiled, slender, organism. The length may vary from 6 to 10 micro meter and width 0.25micro meter. It is better appreciated through dark ground microscope than light microscope due to its narrow morphology and low content of protoplasm .This organism reproduces by binary fission and shows a wide range of movements(motility) ranging from angulation, rotation, buckling, propulsion, expansion, undulation to coil compression.

**Figure 4: Dark ground microscopic appearance of treponemapallidum**



## CLASSIFICATION OF SYPHILIS




## ROUTES OF TRANSMISSION

1. The primary mode by which T.pallidum is transmitted by sexual contact. Although majority population practice heterosexual method , oral sexual contact is emerging as an important mode in MSM.
2. Vertical transmission
3. Needle prick injury in health workers
4. Breast feeding in the presence of infected lesion on breast
5. Kissing[50]
6. Syphilis brephotrophica[51] – Syphilis transmitted to persons , while taking care of babies

## PATHOGENESIS

The organism enters human tissue under favourable conditions via minor abrasion, through sexual contact after which they reach bloodstream and lymphatics as early as few hours after inoculation,[52] where they multiply and rapidly disseminate to other regions. Once multiplication has reached required density, it elicits a local reaction to form chancre , which resolves spontaneously without even treatment by 3 to 8 weeks. After a period of about 2 to 12 weeks features of secondary Syphilis appears which may also heal without treatment in about 2 to 12 weeks. The patient soon enters a asymptomatic period of early latent Syphilis (< 1years) and late latent Syphilis (> 2years). One third of patients after a quiet long time may manifest with features of cardiovascular Syphilis (by 10 – 40 years) and neuro Syphilis (by 3 – 35 years)

## CLINICAL FEATURES

### Primary syphilis

The characterstic feature is Hunterian chancre which is usually a solitary well defined ulcer, round to oval in shape with a button like induration, rolled out edges and floor may be covered with greyish slough or rarely with haemorrhagic slough. It appears after about 3 to 90 days of exposure to organism and the most common site in males is coronal

sulcus followed by glans penis and in females vagina and cervix. Extra genital sites include breast , lips, finger and anorectal sites. Various types of chancre are condom chancre , chancre redux(infectious), pseudochancre redux (non infectious) and Syphilis d emblee, phagedenic chancre, and chancre gallius.

**Secondary Syphilis**

It presents with a wide range of polymorphic lesion ranging from macule, papule, pustule to papulosquamous lesion.

Macular syphilide–appears as pinkish rounded discrete lesion most common on chest, shoulder, and back.

Papularsyphilide – is the characterstic lesion of Secondary Syphilis it is less than 1cm in size and symmetrically distributed on trunk, arms, face, and extremities other forms of papularsyphilide include corona veneris, condylomatalata , annular syphilide , follicularsyphilide (moth eaten alopecia).

Papulosquamoussyphilide – papules covered with predominant scales resembling psoriasiform lesion.

Pustularsyphilide - occurs rarely seen in debilitating patients.

**Mucous membrane involvement**

Oral mucosa – commonly affected sites are mucosa of lip , tongue , pharynx , tonsil ,larynx . Snail track ulcer often appear on tonsil but may also involve other sites.

Genital mucosa – vulval and vaginal mucosa in females and glans penis mucosain males are commonly involved.

Luis maligna[53,54] - Luis maligna initially presents with constitutional symptoms like malaise, fever, arthalgia, headache and photophobia followed by a papulopustular lesion which undergoes necrosis and edges are sharply marginated with thickened rupoid crust.

**Late benign gumma**

Late benign gumma typically presents with punched out ulcer covered with wash leather slough involving palms, soles, mucous membrane of palate, nasal septum, pharynx and also involves internal organs like liver and spleen.

**Cardiovascular Syphilis**

With the effective antibiotic use the patient reaching to cardiovascular Syphilis is becoming rare even in AID's patient and when present it shows a vivid manifestation like myocarditis, aortic regurgitation, aortic aneurysm, coronary artery stenosis and aortitis.

**Neurosyphilis**

Neurosyphilis takes many decades of about 5-35 years for its clinical manifestation and the possible presentation include acute syphilitic meningitis, meningovascular Syphilis, in 10% of cases General Paresis of Insane(GPI) and rarely can present with tabesdorsalis.

**Laboratory diagnosis of Syphilis**

**Primary Syphilis**

Primary Syphilis is diagnosed mainly by microscopic examination of T.pallidum through:

a) Dark Field Microscopy (DFM)

b) Direct Fluorescent Antibody-TreponemaPallidum (DFA-TP)

Serological test are not much reliable since the T. Pallidum can be detected only after a period of 1 to 4 weeks of chancre formation.

**Secondary Syphilis**

Secondary Syphilisis diagnosed by serological tests with a sensitivity of 100% and they are of two types:

- Non-Treponemal tests
- Treponemal tests

- **Non-Treponemal tests**

**a) VDRL**

VDRL in a titre of > 1:8 in a patient with first episode of Syphilis and a four fold elevated titre in a patient with past history of Syphilis indicates a positive result. In cases of patient showing < 1:8 titre, the test should be repeated after a period of 10 weeks or confirmed by a Treponemal test.

**b) Rapid Plasma Reagin (RPR) test**

This test uses cards unlike the slides used inVDRL test and a modifiacation of this test is called TRUST ( toludine red unheated serum test )

- **Treponemal tests**

a) Fluorescent Treponemal antibody absorption test (FTA-Abs)

b) Treponemapallidum immobilization test (TPI)

c) Treponemapallidumhaemagglutination test (TPHA)

d) Microhaemagglutination test for Treponemapallidum (MHA-TP)

**Latent Syphilis**

Since the lesions are absent during this stage definite diagnosis is quiet difficult early latent Syphilis is diagnosed by Non-Treponemal tests however in late latent Syphilis Non-Treponemal tests are negative in 30% cases and such patients results should be confirmed by Treponemal tests.

**Tertiary Syphilis**

Treponemal tests are test are used to diagnose this stage but around 2 to 4% of patients show reports with Treponemal tests and such patients may require other test like PCR.

**Neurosyphilis**

The VDRL-CSF test is considered to be the standard serologic test for neurosyphilis and when reactive in the absence of contamination of the CSF with blood, it is considered diagnostic of neurosyphilis.

**Congenital Syphilis**

Congenital Syphilis can be diagnosed by isolation of T. Pallidum from the nasal discharge or from the umbilical vein blood, but these are not confirmatory test since it can be due to transfer of maternal antibodies.

Hence the diagnostic test include:

a) FTA-ABS test using fluorescent-labelled anti-human globulin.
b) PCR from cerebrospinal fluid.

**Treatment of Syphilis**

**1. Primary and early latent Syphilis**

Injection benzathine penicillin G 2.4 million units given intramuscularly in a single dose with 1.2million units into each gluteal region.

In patients allergic to penicillin alternatively the following drugs can be given

c) Capsule doxycycline 100 mg orally twice daily for 14 days, or
d) Capsule tetracycline 500 mg orally 4 times daily for 14 day

**2. Late latent Syphilis,Gumma and cardiovascular Syphilis**

Injection benzathine penicillin G 7.2 million units total, which is administered as 3 doses of 2.4 million units intramuscularly each at 3-week intervals.

In patients allergic to penicillin alternatively the following drugs can be given.

a) Capsule doxycycline 100 mg orally twice daily for 30 days OR

b) Capsule tetracycline 500 mg orally 4 times daily for 30 days.

**3.Neurosyphilis**

Injection aqueous crystalline penicillin G 18-24 million units per day, administered as 3-4 million units IV every 4 hours or by continuous infusion for 10-14 days given intravenously .

In patients allergic to penicillin alternatively the following drugs can be given Injection procaine penicillin 2.4 million units IM once daily along with tablet Probenecid 500 mg orally 4 times a day, both given for 10-14 days.

**4. Congenital Syphilis**

Injection aqueous crystalline penicillin G 1 to 1.5 lakh units/kg/day given in a divided dose 50,000units/kg/dose through intravenous route for every 12 hours for the intial 7 days which is then given $8^{th}$ hourly for a total period of 10 days. Alternatively injection procaine penicillin 50,000units/kg/dose intramuscularly once daily for 10 days can be given.

**Hepatitis B and Hepatitis C**

Among the viral causes of infectious Hepatitis, most common and major health problems are Hepatitis B and Hepatitis C. Greater than one

third population in the world are documented to be Hepatitis B and Hepatitis C infected. Though HBV(double stranded DNA virus) and HCV(single stranded RNA virus) are structurally different they share similar routes of transmission and clinical presentation.

**Routes of transmission**

Hepatitis B and Hepatitis C are predominately transmitted through blood transfusion and blood products[55] other possible modes of transmission include

- Venereal route[56]
- Vertical transmission[57]
- Intravenous drug abuse[58]
- Infected needles and syringes[59]
- Saliva[60]
- Tatooing[61]

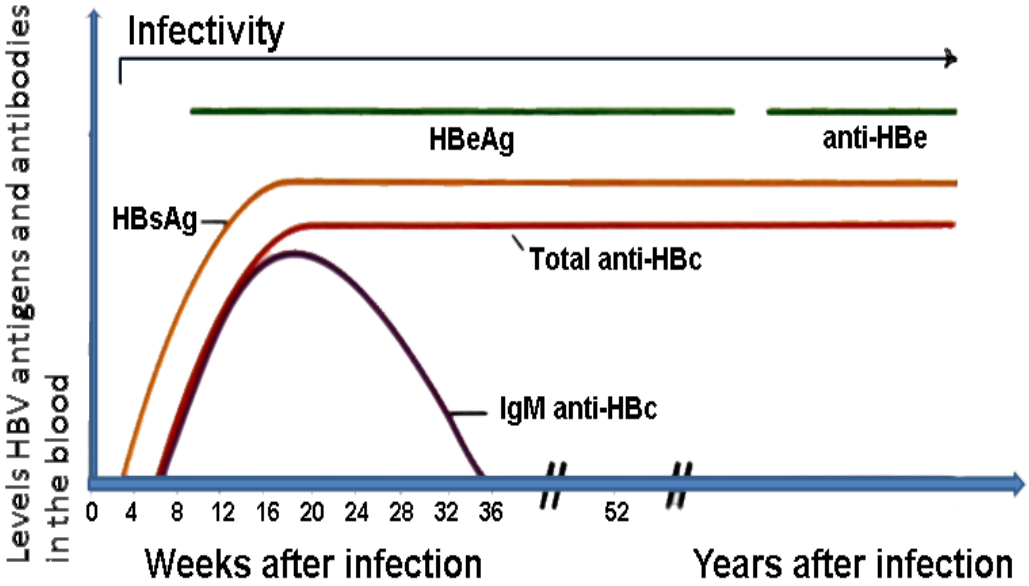
**Clinical features**

Incubation period of Hepatitis B vary from 1 to 6 months compared to 15 to 150 days of Hepatitis C. Clinically both HBV and HCV intially presents with constitutional symptoms like fatigue, anorexia, malaise, myalgia, nausea vomiting, arthalgia, and less frequently they can present with headache, pharyngitis, cough,

photophobia and coryza 1 to 2 week before the onset of jaundice. These prodromal features reduces with onset of jaundice.

During the icteric phase patients can present with upper abdominal pain due to liver and spleen enlargement, cervical lymphadenopathy. Acute Hepatitis B infection in 90-95% normally recovers within 1-2 months whereas about 1-10% of cases remain chronically infected and can go for cirrhosis or persist as chronic liver disease.[8]Similarly Hepatitis C in 1% cases can enter into phase of fulminant Hepatitis or progress to hepatocellular carcinoma.

**Figure 5: Various antigens and antibodies demonstrable in HBV infection**



# Hepatitis C Virus Infection

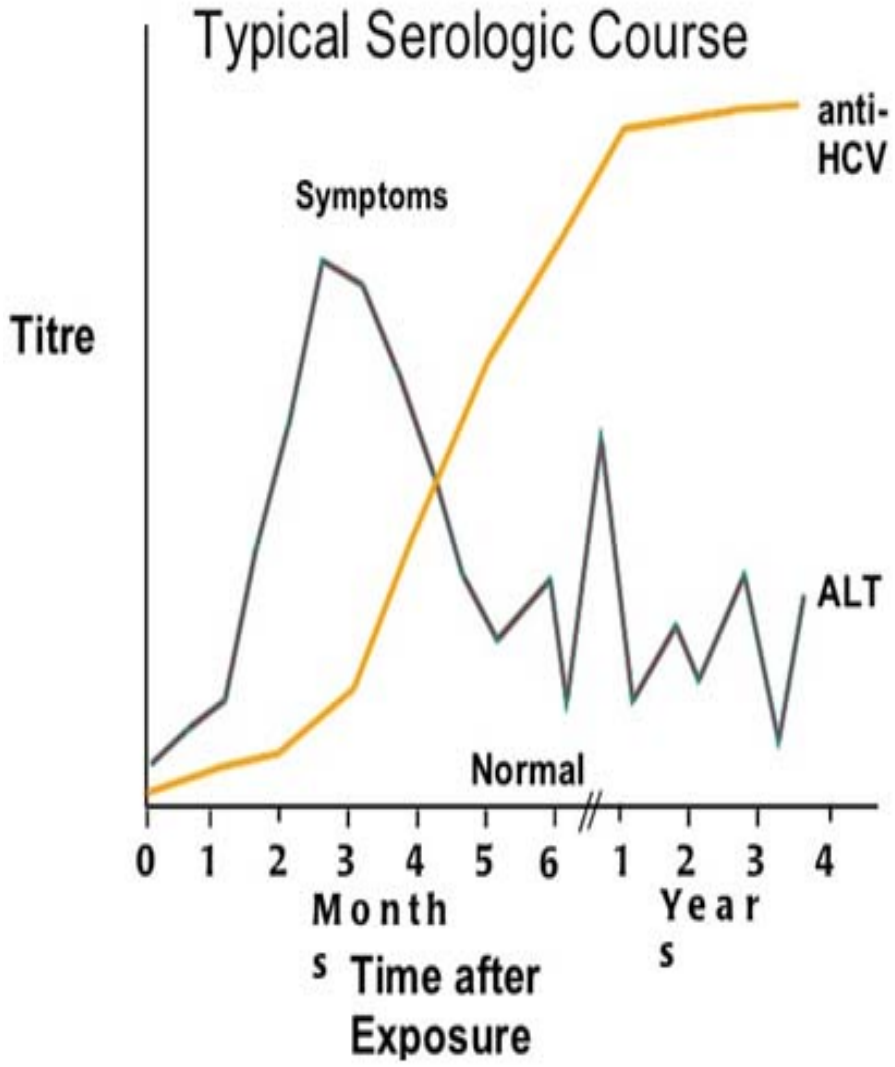


**Figure 6 : HBsAg ELISA plate showing positive and negative controls**

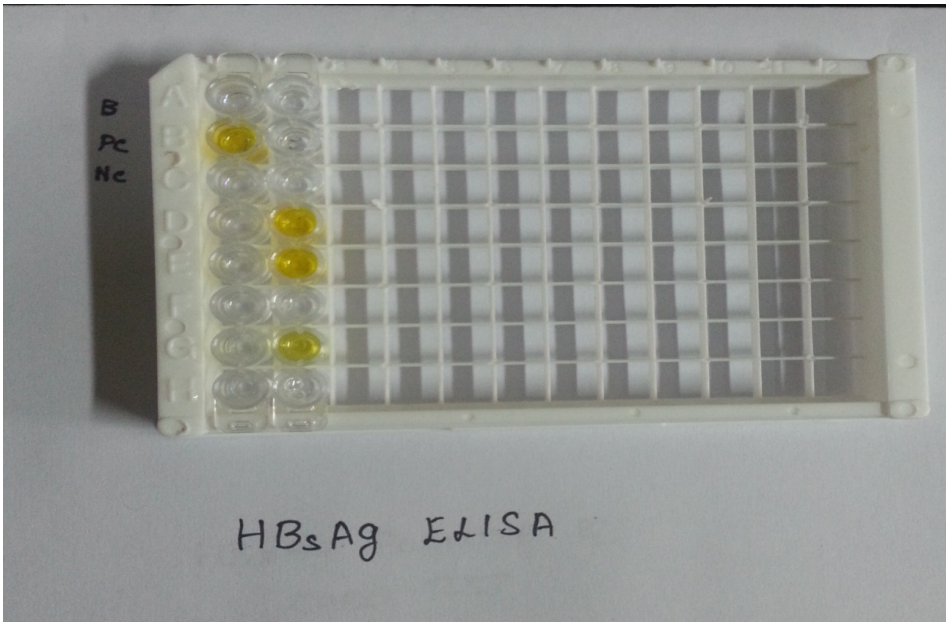



**Figure 7:HBsAg positive by card test method**

# *Aims & Objectives*

# AIMS & OBJECTIVES

1. To detect the Prevalence of Hepatitis B, Hepatitis C and Syphilis and Detection of Liver enzymes levels in HIV positive patients.
2. To establish the link of Liver enzyme levels by LFT in Hepatitis infection and HIV positive patients.
3. To study the Liver enzymes levels in patients who are VDRL reactive and HIV positive.

# *Materials and Methods*

# MATERIALS & METHODS

## STUDY DESIGN

Prospective Observational study

## STUDY GROUP

100 HIV positivepatients attending the STI Out Patient Department, Institute of Venereology, Madras Medical College / RGGGH, Chennai are selected randomly.

The Institute ethics committee clearance was obtained and informed consent was taken from all the patients included in study group.

## INCLUSION CRITERIA

All HIV positive patients attending STI Out Patient Department with High Risk like:

1. Female sex workers
2. Partners of HIV seropositive / VDRL reactive patients
3. Transgenders
4. Homosexuals
5. Victims of sexual abuse
6. Intravenous drug abusers
7. H/o multiple blood transfusions
8. Occupational exposure to blood products

## EXCLUSION CRITERIA

1. Patients who refuse to participate in the study
2. Children and Patients above 60 years

## PROCEDURE

A detailed and thorough history was obtained pertaining to the following parameters:

- Age
- Occupation
- Socioeconomic status
- Marital and obstetric history
- Sexual history
- History of blood transfusion
- History related to sexually transmitted infections as per the proforma enclosed.
- Past, Personal, Treatment history shall be documented along with clinical examination.

Under aseptic precautions, about 5ml of blood is withdrawn from a vein and centrifuged by 2500 RPM to separate the serum, the sample is then tested for HIV by Rapid test (dot immunoassay) kit, wherein the immobilized antigen-antibody complex is visualized by means of colour

producing (chromogenic) reaction. The coloured endpoint is developed by Colloidal Gold –Protein –A Signal Reagent.10 patients' blood samples who are HIV negative was taken as controls.

HIV positive blood samples are then subjected to:

**1. VDRL test**

The centrifuged serum is inactivated by keeping in hot water bath for 30min at 56$^{o}$C the test is usually carried out qualitatively and if qualitative test shows positivity it is subjected to quantitative test.

**Qualitative test**

In qualitative test 0.05ml of serum is added to one drop of antigen. The mixture is then rotated for 4min at 180RPM in a VDRL rotator, along with this positive and negative control samples will be tested, the wells are then observed through microscope to detect any flocculation reaction and reported as follows:

1. No clumps or slight roughness-Non Reactive
2. Small clumps-Weakly positive
3. Medium and large clumps- Reactive

**Quantitative test**

Reactive cases are then subjected to quantitative test which is done by slide flocculation or tube flocculation test.

**Slide flocculation test:**

- 0.5ml of normal saline is pipetted out in each well of slide starting from 2$^{nd}$ well.
- In well 1, 0.5ml of undiluted serum is taken
- 0.5ml of test serum is pipetted out in well in 1:2 dilution
- 0.5ml from well 2 is taken and added to well 3, like this1:4 dilution continued upto 1:64
- Positive and negative controls for each test is incorporated
- 1 drop of viral antigen is added to each ring with 18 gauge needle
- The wells are then rotated for 4min at 180RPM and seen under microscope and the highest dilution that has flocculation is reported reactive titre.

**2. HBsAg detection using HEPALISA kit**

HEPALISA is a solid phase enzyme linked immunosorbentassay (ELISA) it is based on the ‘’Direct Sandwich’’ principle.

Here the microwells are coated with monoclonal antibodies with high reactivity for HBsAg.The samples are added in the wells followed

by addition of enzyme conjugate linked to Horseradish Peroxidase(HPRO).A sandwich complex is formed in the well wherein HBsAg (from serum sample) is ‘’trapped’’ or ‘’sandwiched’’ between the antigen and antibody HPRO conjugate. Unbound conjugate is then washed off with wash buffer. The amount of bound peroxidase is proportional to the concentration of HBsAg present in the sample.

Upon addition of substrate buffer and chromogen, a blue colour develops. The intensity of blue colour developed is proportional to the concentration of HBsAg in sample.To limit the enzyme-substrate reaction, stop solution(sulfuric acid) is added and a yellow colour develops which is finally read at 450nm spectrophotometrically.

**Figure 8: Contents of HEPALISA kit**

### 3. Detection of anti-HCV antibody by using 3rd generation HCV Microlisa kit

Diluted sample and controls are incubated for 30min.Antibodies to HCV, if present, bind to immobilized HCV antigens on the microwell.

The micro wells are then thoroughly washed with the diluted wash buffer to remove excess of unbound anti-HCV or other human IgGs which may interfere with the test. An enzyme conjugate anti-human IgG conjugated with HPRO is added.

The enzyme substrate reaction leads to development of blue colour which is indicative of Ag-Ab reaction.Finally, the stop solution is added and the optical density of the developed colour is read photometrically.

**Figure 9: Contents of HCV microlisa kit**

# *Observation and Results*

# OBSERVATION AND RESULTS

## SEX DISTRIBUTION OF HIV POSITIVE PATIENTS

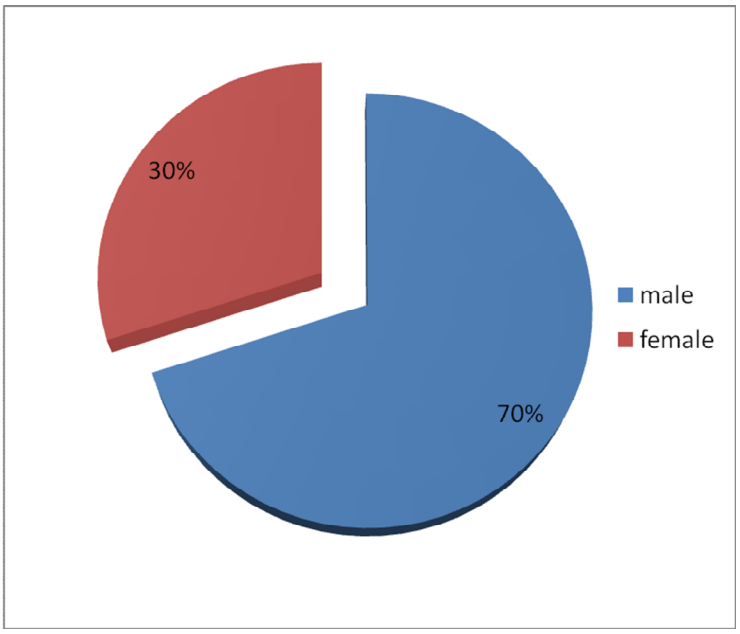
100 HIV positive patients fulfilling the inclusion criteria were enrolled in the study .

Out of these 100 patients, 70(70%) were males and 30(30%) were females. Thus, HIV infection was more common in males than females with male:female ratio as 7:3.

**Table 1 : SEX DISTRIBUTION (N=100)**

| MALE | FEMALE |
|---|---|
| **70** | **30** |

**Figure 1 : SEX DISTRIBUTION**



## AGE DISTRIBUTION OF HIV POSITIVE PATIENTS

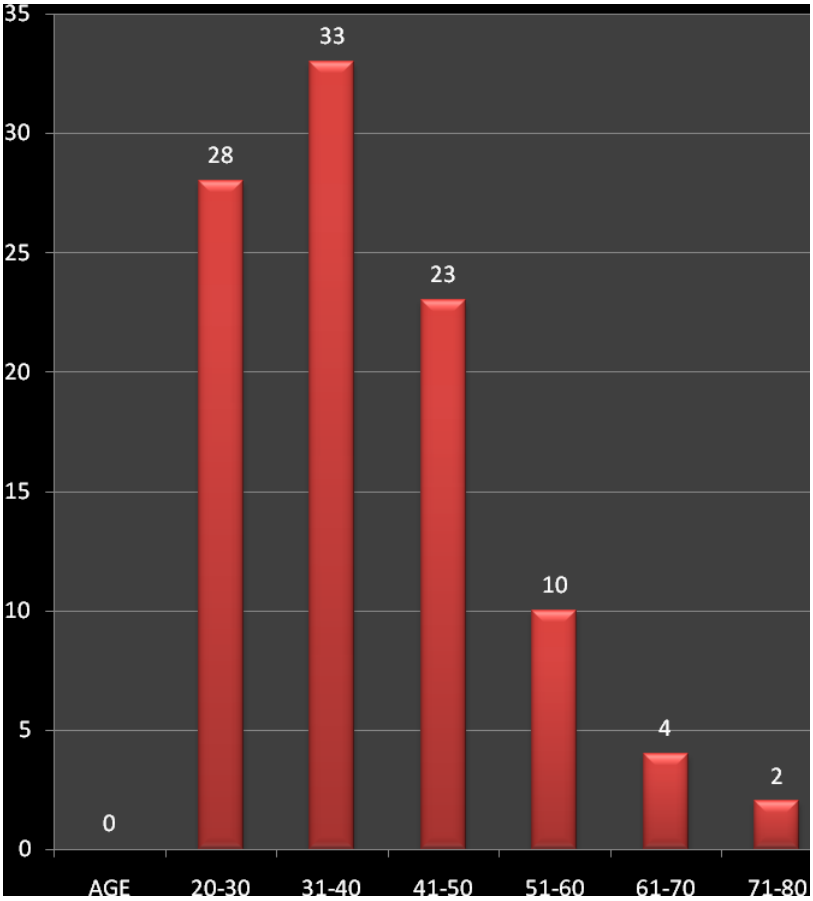
The age of the patients ranged from 20 to 80 years. The most commonly affected age group was 31 – 40 years with 33 (33%) patients, closely followed by 20-30 years age group with 28 (28%) patients .

Thus HIV infection was most common in the age group of 31-40years.

**Table 2: AGE DISTRIBUTION**

| **AGE** | **n=100** |
|---|---|
| 20-30 | 28 |
| 31-40 | 33 |
| 41-50 | 23 |
| 51-60 | 10 |
| 61-70 | 4 |
| 71-80 | 2 |
| **TOTAL** | **100** |

**Figure:2 AGE DISTRIBUTION OF HIV POSITIVE PATIENTS**



## SEX WISE AGE DISTRIBUTION OF HIV POSITIVE PATIENTS

Maximum number of affected males were in the age group of 31-40years With 22 (34.1%) males in this group .

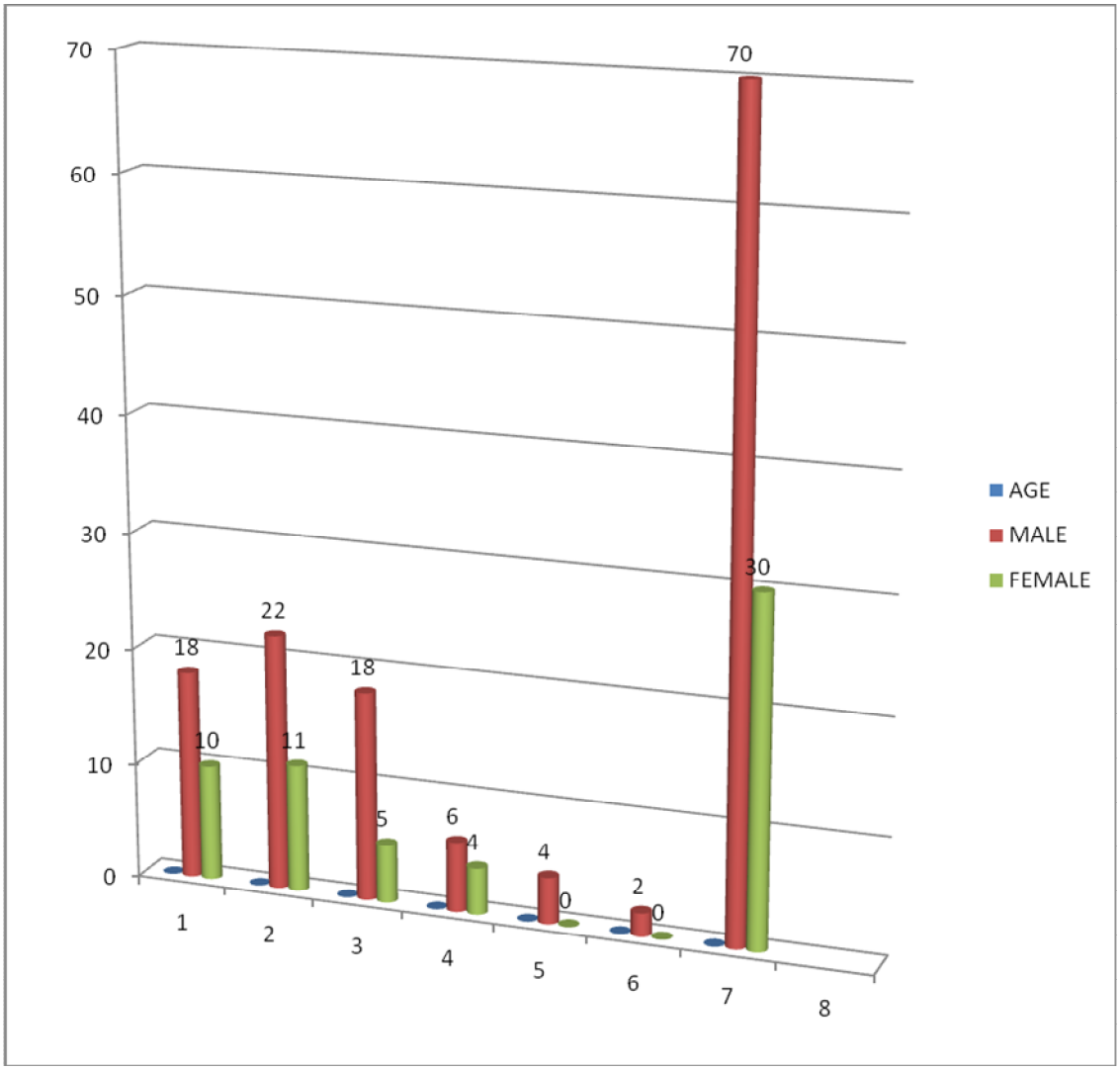
It was followed by 20-30 years with 18(25.7%) males and 41 to 50 years with 18(25.7%) males respectively.

Females were also affected more in the age group of 31to 40 years with a total of 11 (36.6%) women in this age group, followed by 41 to 50 years group with 10 (33.3%) patients(Figure 11).

**Table 3: SEX WISE AGE DISTRIBUTION**

| AGE | MALE | FEMALE |
|---|---|---|
| 20-30 | 18 | 10 |
| 31-40 | 22 | 11 |
| 41-50 | 18 | 5 |
| 51-60 | 6 | 4 |
| 61-70 | 4 | 0 |
| 71-80 | 2 | 0 |
| **TOTAL** | **70** | **30** |

**Figure: 3 SEX WISE AGE DISTRIBUTION OF HIV POSITIVE PATIENTS**



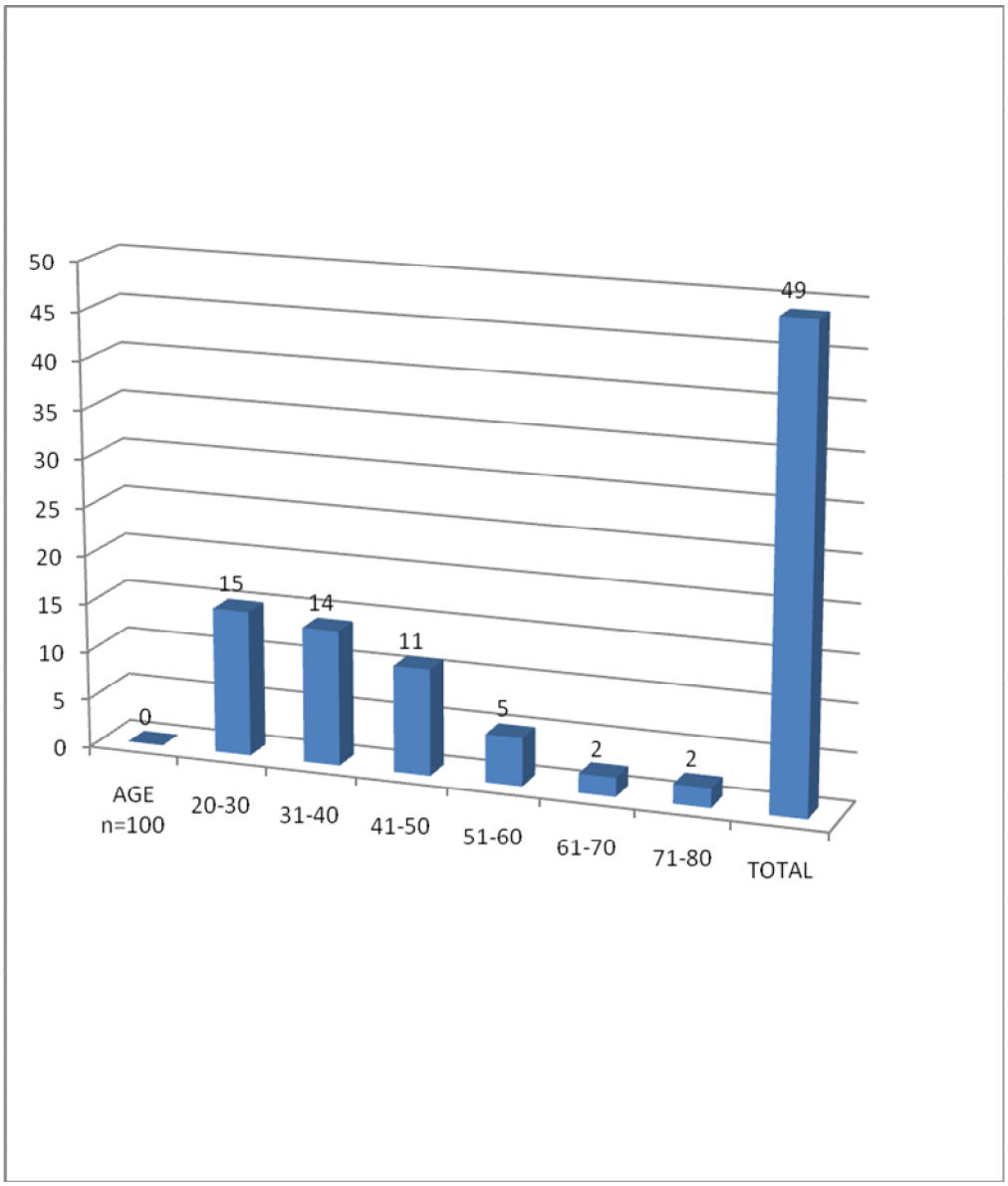
**DISTRIBUTION OF ALCOHOLICS IN HIV POSITIVE PATIENTS**

Out of the 70 male patients, 49(70%) were alcoholics and the most common affected age group were 20-30 years with 15(21.4%) patients followed by 31-40 years with 14(20%) patients.

**Table 4: DISTRIBUTION OF ALCOHOLICS IN HIV POSITIVE PATIENTS**

| **AGE** | **n=49** |
|---|---|
| 20-30 | 15 |
| 31-40 | 14 |
| 41-50 | 11 |
| 51-60 | 5 |
| 61-70 | 2 |
| 71-80 | 2 |
| **TOTAL** | **49** |

**Figure 4: DISTRIBUTION OF ALCHOLICS IN HIV POSITIVE PATIENTS**



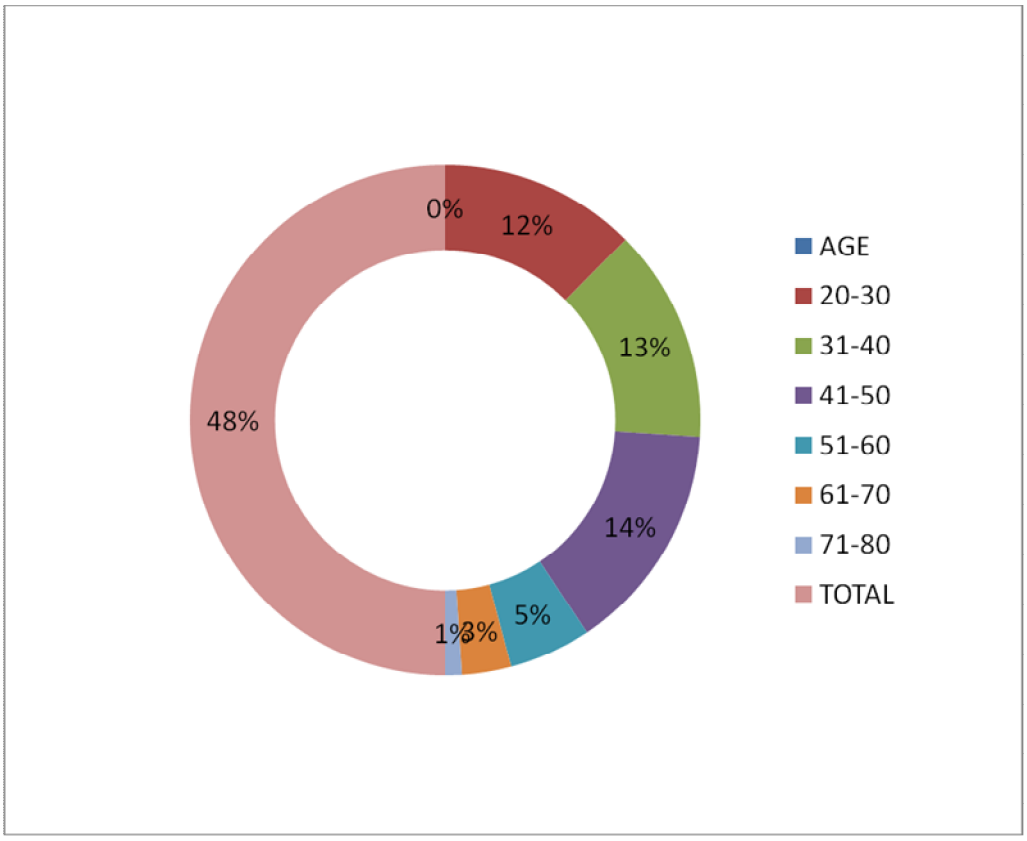
**DISTRIBUTION OF SMOKERS IN HIV POSITIVE PATIENTS**

Among the 70 male patients, 48(68.5%) were smokers and the most common affected age group were 31-40 years with 13(18.5%) patients followed by 20-30years with 12(17.1%) patients.

**Table 5**: **DISTRIBUTION OF SMOKERS IN HIV POSITIVE PATIENTS**

| **AGE** | **n=48** |
|---|---|
| 20-30 | 12 |
| 31-40 | 13 |
| 41-50 | 14 |
| 51-60 | 5 |
| 61-70 | 3 |
| 71-80 | 1 |
| **TOTAL** | **48** |

**Figure 5: DISTRIBUTION OF SMOKERS IN HIV POSITIVE PATIENTS**



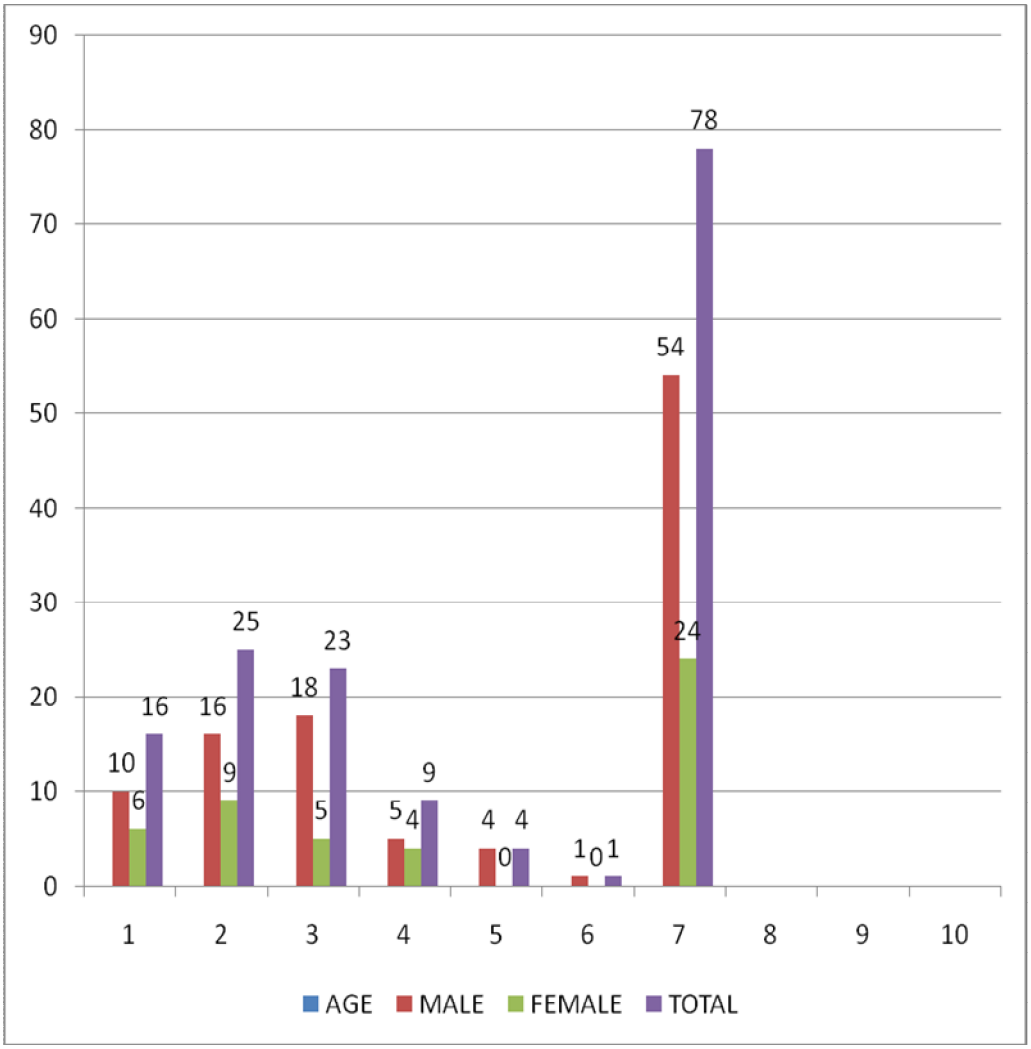
## MARITAL STATUS IN HIV POSITIVE PATIENTS

Out of 100 patients, 78 were married and among these the most common affected age group was 31-40 years with 25(25%) patients followed by 41-50 years with 2(23%) patients.

**Table 6: MARITAL STATUS IN HIV POSITIVE PATIENTS**

| AGE | MALE | FEMALE | TOTAL |
|---|---|---|---|
| 20-30 | 10 | 6 | 16 |
| 31-40 | 16 | 9 | 25 |
| 41-50 | 18 | 5 | 23 |
| 51-60 | 5 | 4 | 9 |
| 61-70 | 4 | 0 | 4 |
| 71-80 | 1 | 0 | 1 |
| **TOTAL** | **54** | **24** | **78** |

**Figure 6: MARITAL STATUS IN HIV POSITIVE PATIENTS**



**ELEVATED LIVER ENZYME LEVELS IN HIV POSITIVE PATIENTS**

Out of the 100 HIV positive patients, liver enzyme was elevated in 4(4%) patients, among these, 3 patients showed elevated SGPT, SGOT and 4 patient showed elevated alkaline phosphatase with ;

SGPT- 60 IU, 76 IU, and 120 IU

SGOT- 80 IU, 68 IU and 94 IU and

Alkaline phosphatase -133 IU, 140 IU, 166 IU and 140 IU

**Table 7: ELEVATED LIVER ENZYME LEVEL IN HIV POSITIVE PATIENTS**

| AGE | No of patients | SGPT (0-40 IU) | SGOT (0-45 IU) | ALKP (30-120 IU) |
|---|---|---|---|---|
| 20-30 | 1 | 76 | 68 | 140 |
| 31-40 | 1 | 120 | 94 | 166 |
| 41-50 | 0 | - | - | - |
| 51-60 | 1 | - | - | 140 |
| 61-70 | 1 | 60 | 80 | 133 |
| 71-80 | 0 | - | - | - |

**Figure 7: ELEVATED LIVER ENZYME LEVEL IN HIV POSITIVE PATIENTS**

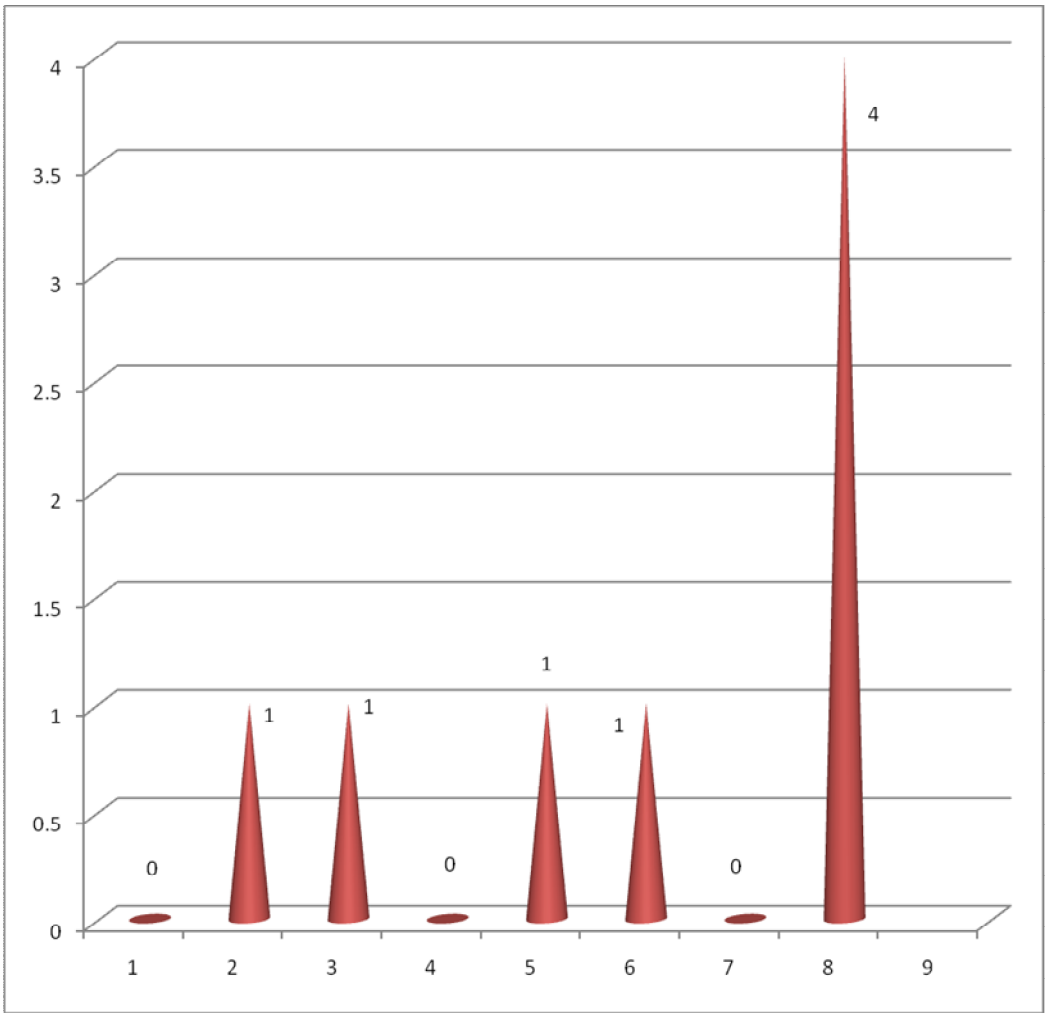
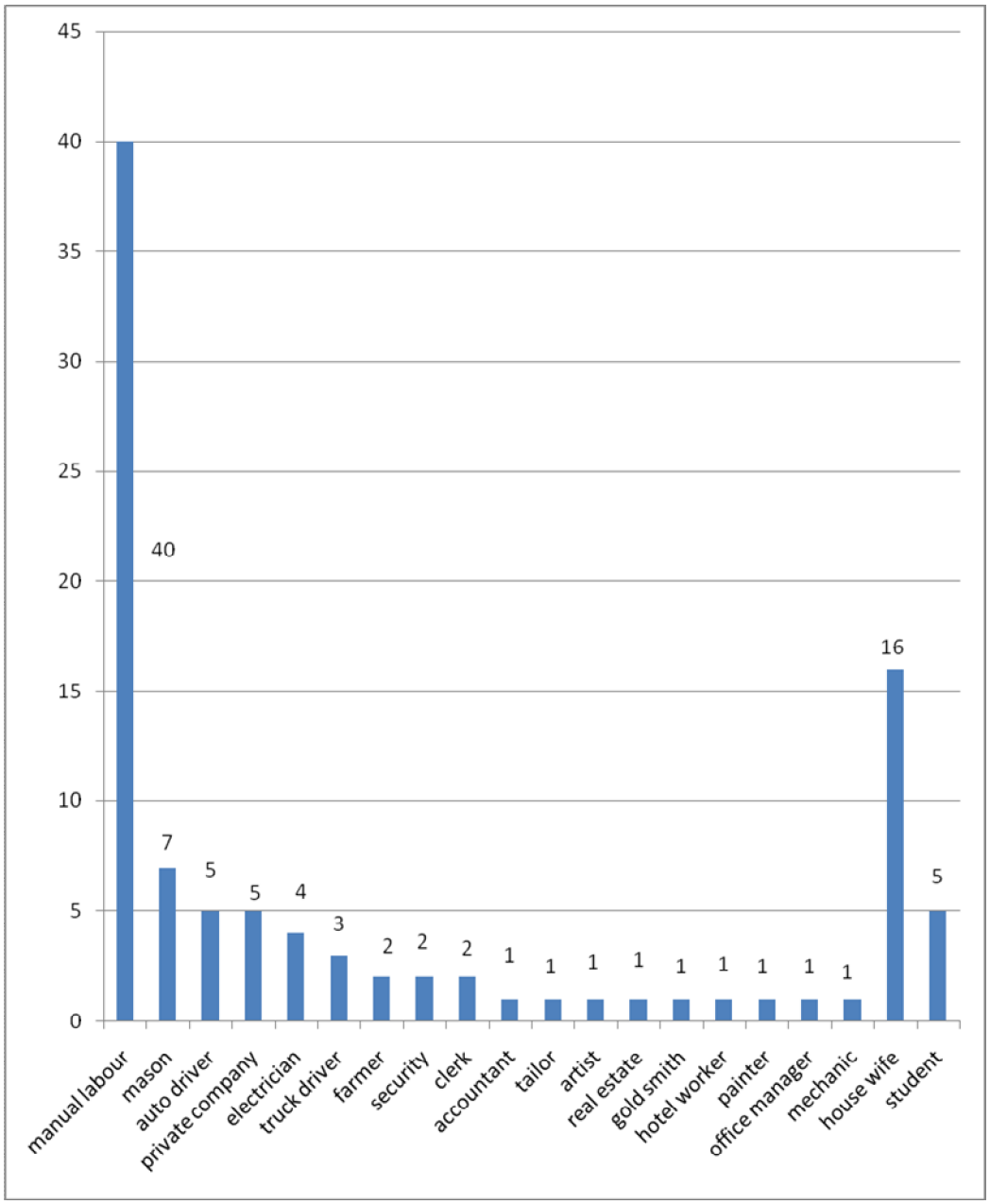


**Table 8 : OCCUPATION OF HIV POSITIVE PATIENTS**

| OCCUPATION | NO OF PATIENTS |
|---|---|
| Manual labor | 40 |
| Mason | 7 |
| Auto driver | 5 |
| Private company | 5 |
| Electrician | 4 |
| Truck driver | 3 |
| Farmer | 2 |
| Security | 2 |
| Clerk | 2 |
| Accountant | 1 |
| Tailor | 1 |
| Artist | 1 |
| Real estate | 1 |
| Gold smith | 1 |
| Hotel worker | 1 |
| Painter | 1 |
| Office manager | 1 |
| Mechanic | 1 |
| House wife | 16 |
| Student | 5 |
| **TOTAL** | **100** |

**Figure 8: OCCUPATION OF HIV POSITIVE PATIENTS**



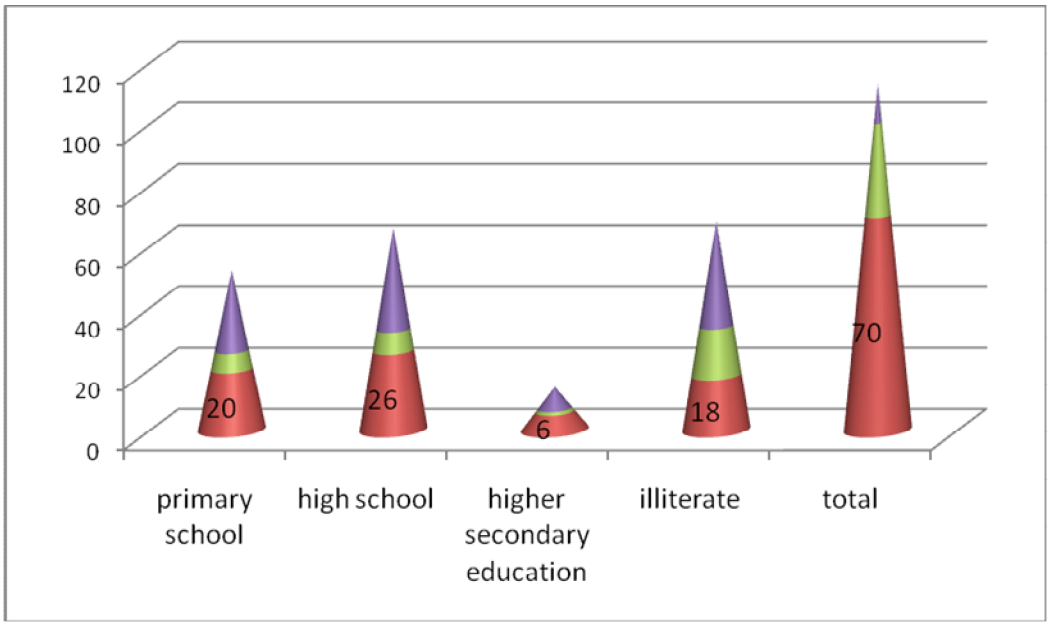
## LITERACY LEVEL IN HIV POSITIVE PATIENTS

Out of 100 patients, 34 were illiterate and the highest level of literacy among others were upto high school (33%) followed by primary school (26%).

**Table 9 : LITERACY LEVEL IN HIV POSITIVE PATIENTS**

| EDUCATION | MALE | FEMALE | TOTAL |
|---|---|---|---|
| Primary school | 20 | 6 | 26 |
| High school | 26 | 7 | 33 |
| Higher secondary education | 6 | 1 | 7 |
| Illiterate | 18 | 16 | 34 |
| **TOTAL** | **70** | **30** | **100** |

**Figure 9 : LITERACY LEVEL IN HIV POSITIVE PATIENTS**



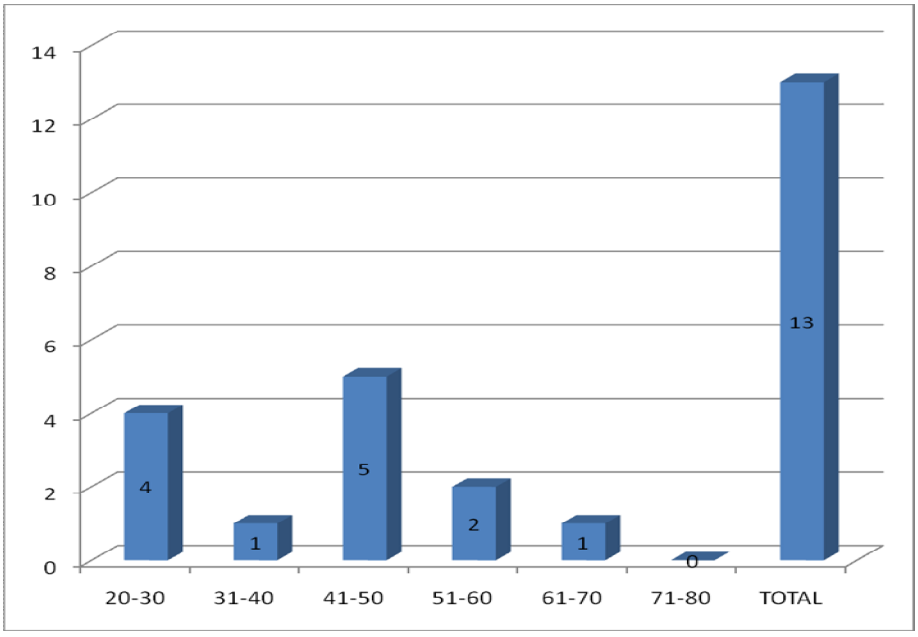
## AGE DISTRIBUTION IN HBsAg POSITIVE HIV PATIENTS

Out of 13 HBsAg positive HIV patients, the maximum number of affected individuals belonged to 41 to 50 years with 5(38.6%) patients followed by 20 to 30 years with 4(30.7%) patients.

**Table 10**: **AGE DISTRIBUTION IN HBsAg POSITIVE HIV PATIENTS**

| AGE | n=13 |
|---|---|
| 20-30 | 4 |
| 31-40 | 1 |
| 41-50 | 5 |
| 51-60 | 2 |
| 61-70 | 1 |
| 71-80 | 0 |
| **TOTAL** | **13** |

**Figure 10: AGE DISTRIBUTION IN HBsAg POSITIVE HIV PATIENTS**



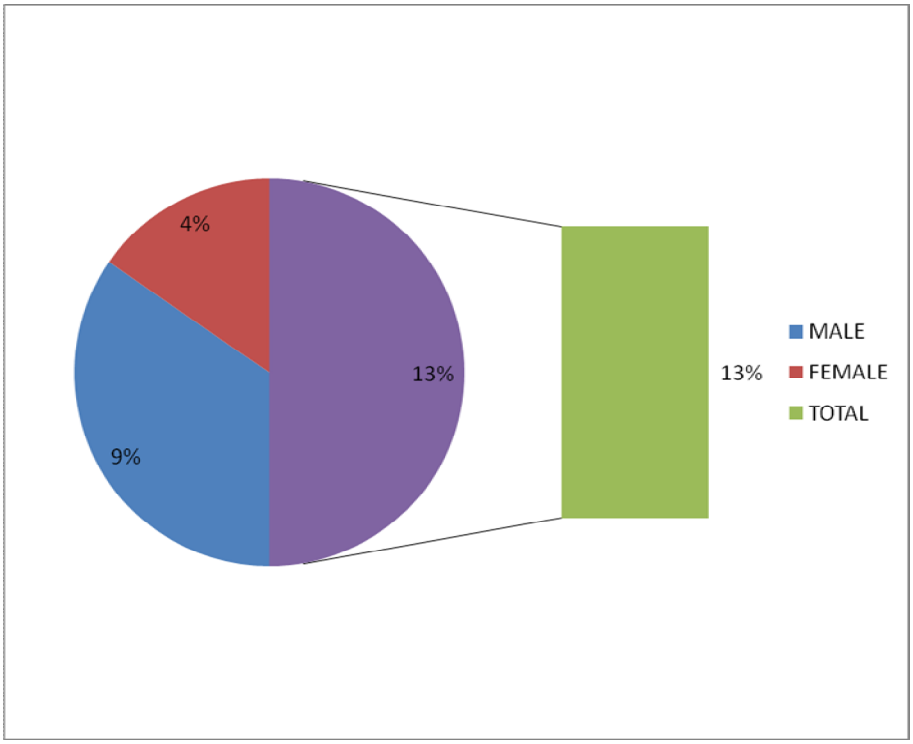
## SEX DISTRIBUTION IN HBsAg POSITIVE HIV PATIENTS

Out of 13 HBsAg positive HIV patients, 9(69.2%) were male and 4(30.7%) were female thus Hepatitis B were more common among male patients.

**Table 11: SEX DISTRIBUTION IN HBsAg POSITIVE HIV PATIENTS**

| MALE | FEMALE | TOTAL |
|---|---|---|
| 9 | 4 | 13 |

**Figure 11: SEX DISTRIBUTION IN HBsAg POSITIVE HIV PATIENTS**



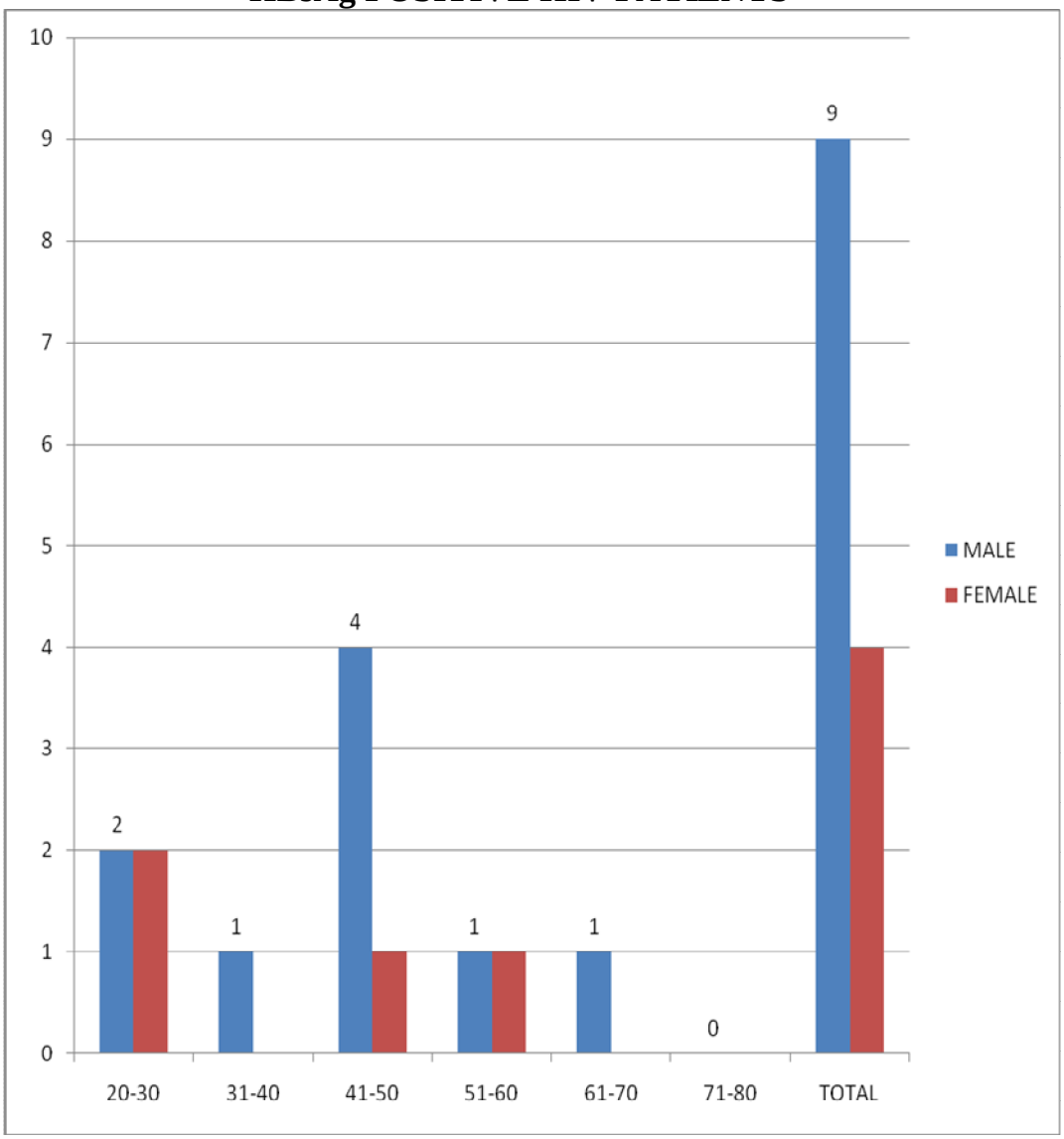
**SEX WISE AGE DISTRIBUTION IN HBsAg POSITIVE HIV PATIENTS**

Out of 13 HBsAg positive HIV patients, the most common affected age group among males was 41-50 years with 4(30.7%) patients and in female was 20 to 30 years with 2(15.8%) patients.

**Table 12: SEX WISE AGE DISTRIBUTION IN HBsAg POSITIVE HIV PATIENTS**

| AGE | MALE | FEMALE |
|---|---|---|
| 20-30 | 2 | 2 |
| 31-40 | 1 | 0 |
| 41-50 | 4 | 1 |
| 51-60 | 1 | 1 |
| 61-70 | 1 | 0 |
| 71-80 | 0 | 0 |
| **TOTAL** | **9** | **4** |

**Figure 12: SEX WISE AGE DISTRIBUTION IN HBsAg POSITIVE HIV PATIENTS**



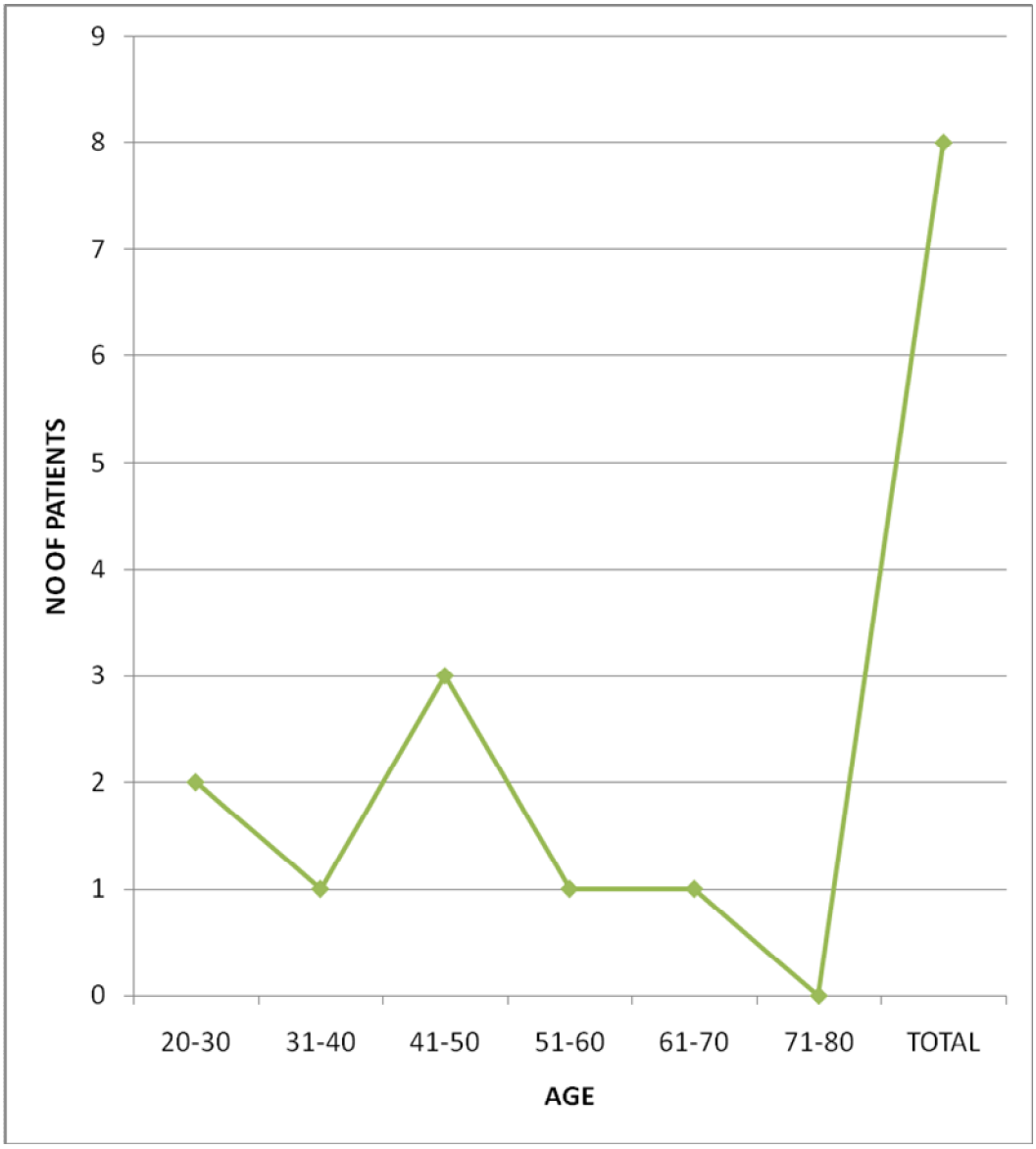
## ALCOHOLIC DISTRIBUTION IN HBsAg POSITIVE HIV PATIENTS

Out of 13 HBsAg positive HIV patients, 8 patients were alcoholics with maximum no of patients in the age group of 41-50 years with 3(23%) patients.

**Table 13: ALCOHOLIC DISTRIBUTION IN HBsAg POSITIVE PATIENTS**

| AGE | n=13 |
|---|---|
| 20-30 | 2 |
| 31-40 | 1 |
| 41-50 | 3 |
| 51-60 | 1 |
| 61-70 | 1 |
| 71-80 | 0 |
| **TOTAL** | **8** |

**Figure 13 : ALCOHOLIC DISTRIBUTION IN HBsAg POSITIVE HIV PATIENTS**



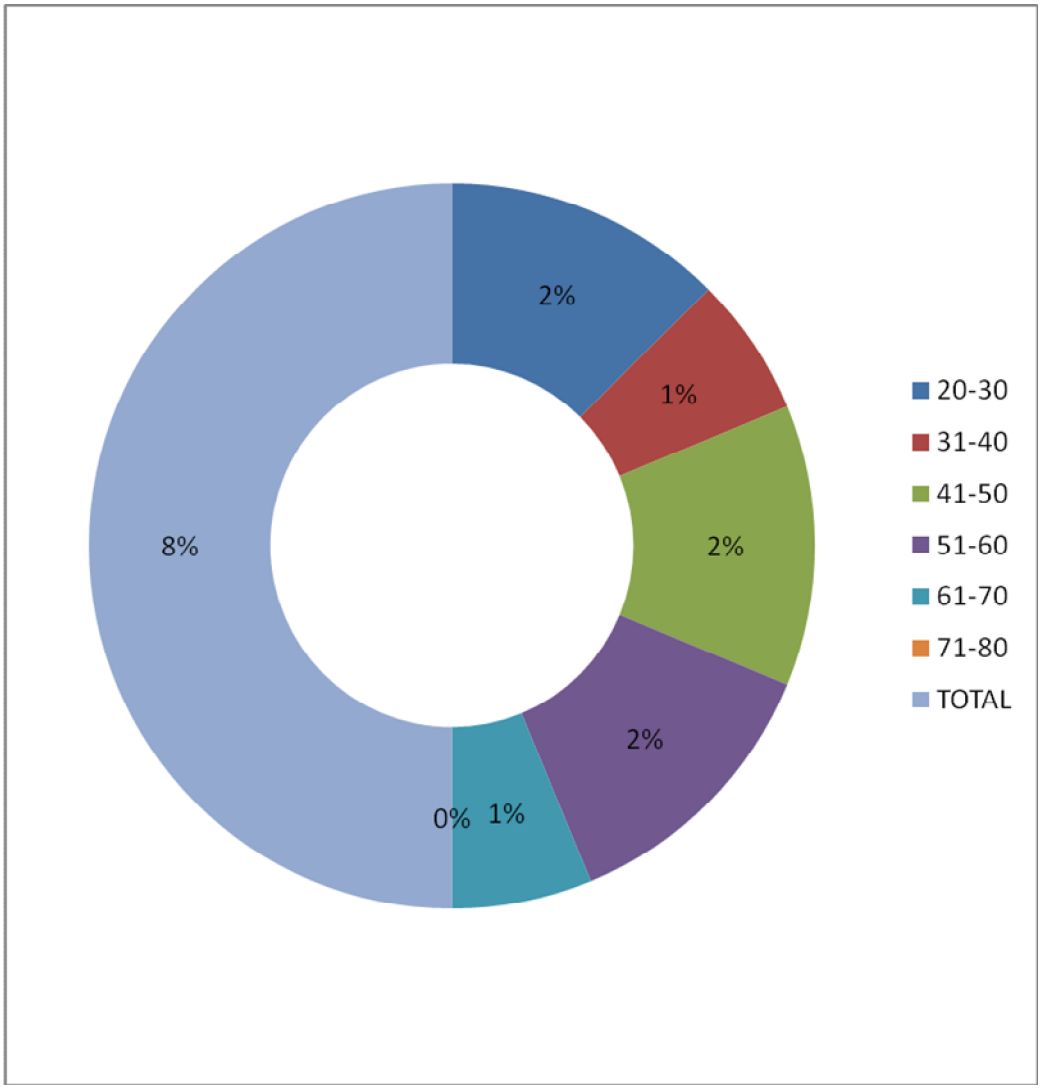
**SMOKERS DISTRIBUTION IN HBsAg POSITIVE HIV PATIENTS**

Out of 13 HBsAg positive HIV patients 8 patients were smokers with maximum no of patients in the age group of 20 to 30 years with 2(25%) patients.

**Table 14: SMOKERS DISTRIBUTION IN HBsAg POSITIVE HIV PATIENTS**

| **AGE** | **n=8** |
|---|---|
| 20-30 | 2 |
| 31-40 | 1 |
| 41-50 | 2 |
| 51-60 | 2 |
| 61-70 | 1 |
| 71-80 | 0 |
| **TOTAL** | **8** |

**Figure14: SMOKERS DISTRIBUTION IN HBsAg POSITIVE HIV PATIENTS**



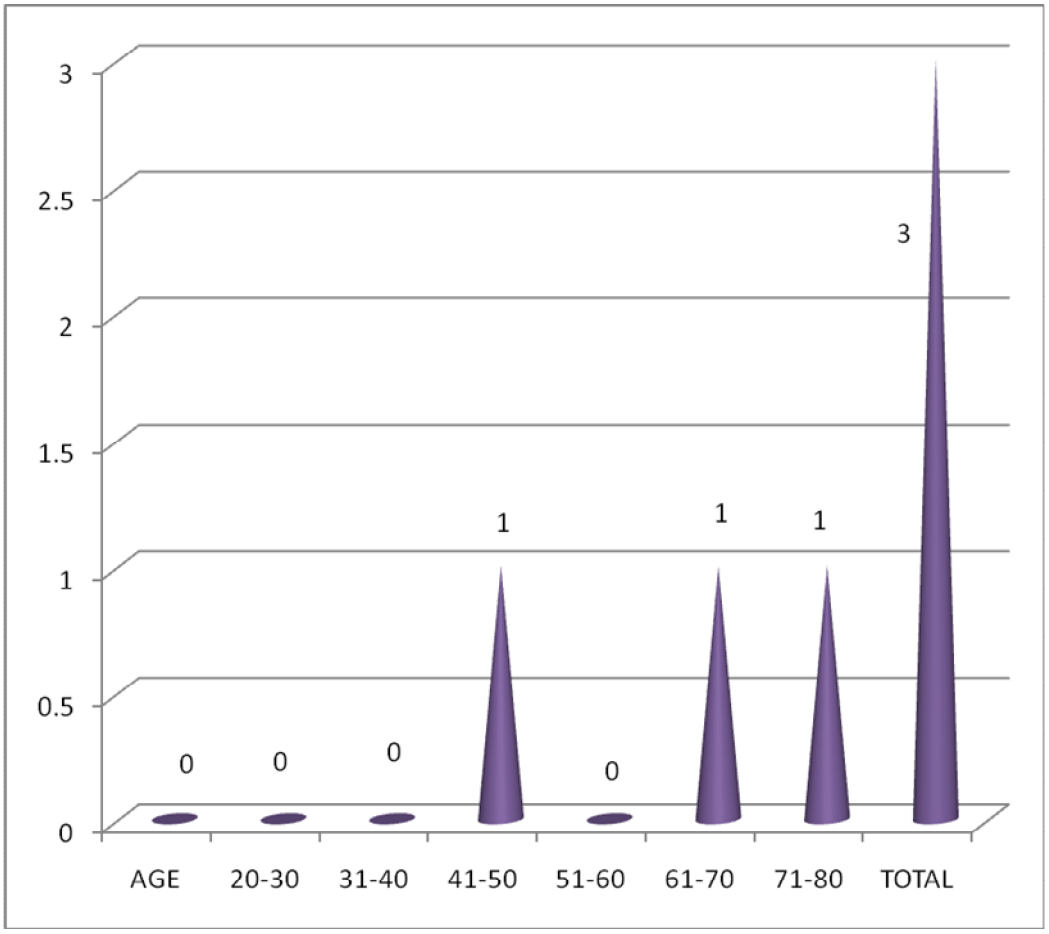
**ELEVATED LIVER ENZYMES IN HBsAg POSITIVE HIV PATIENTS**

Out of 13 HBsAg positive HIV patients, elevated liver enzymes were found in 3(23%) patients. All these 3 patients showed elevated SGPT (with 60 IU, 76 IU, and 120 IU), SGOT (with 80 IU, 68 IU and 94 IU) and alkaline phosphatase(with 133,140 and 166 IU) and they belonged to $2^{nd}$, $3^{rd}$ and $6^{th}$ decade.

**Table 15: ELEVATED LIVER ENZYMES IN HBsAg POSITIVE PATIENTS**

| AGE | No of patients | SGPT (0-40 IU) | SGOT (0-45 IU) | ALKP (30-120 IU) |
|---|---|---|---|---|
| 20-30 | 1 | 76 | 68 | 140 |
| 31-40 | 1 | 120 | 94 | 166 |
| 41-50 | 0 | - | - | - |
| 51-60 | 1 | - | - | - |
| 61-70 | 1 | 60 | 80 | 133 |
| 71-80 | 0 | - | - | - |

**Figure 15: ELEVATED LIVER ENZYMES IN HBsAg POSITIVE HIV PATIENTS**



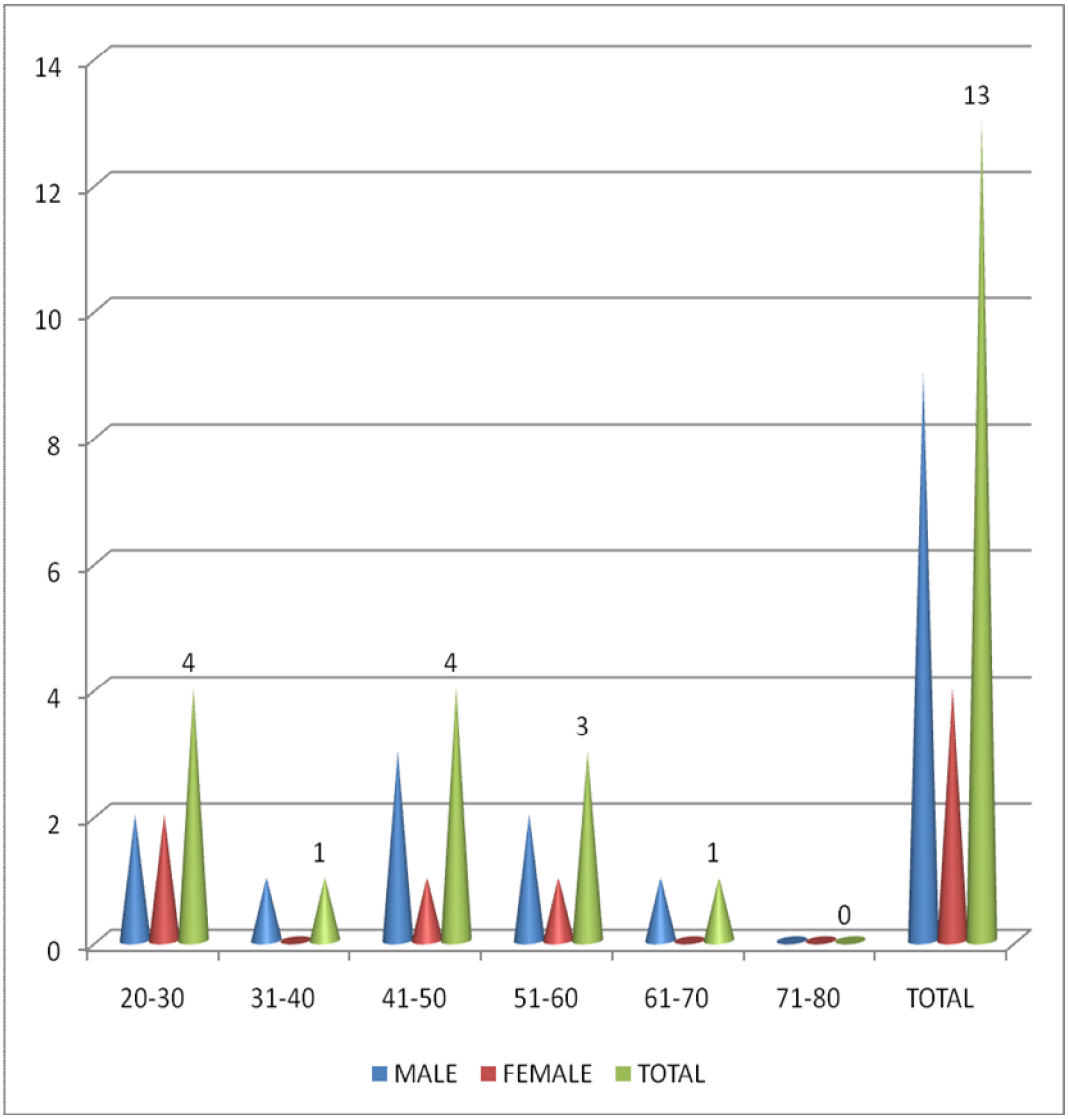
## MARITAL STATUS IN HBsAg POSITIVE HIV PATIENTS

Out of 13 HBsAg positive HIV patients, married individuals were maximum in the age group of 20 -30 years with 4(30.7%) patients and 41-50 years with 4(30.7%) patients.

**Table 16 : MARITAL STATUS IN HBsAg POSITIVE HIV PATIENTS**

| AGE | MALE | FEMALE | TOTAL |
|---|---|---|---|
| 20-30 | 2 | 2 | 4 |
| 31-40 | 1 | 0 | 1 |
| 41-50 | 3 | 1 | 4 |
| 51-60 | 2 | 1 | 3 |
| 61-70 | 1 | 0 | 1 |
| 71-80 | 0 | 0 | 0 |
| **TOTAL** | **9** | **4** | **13** |

**Figure 16: MARITAL STATUS IN HBsAg POSITIVE HIV PATIENTS**



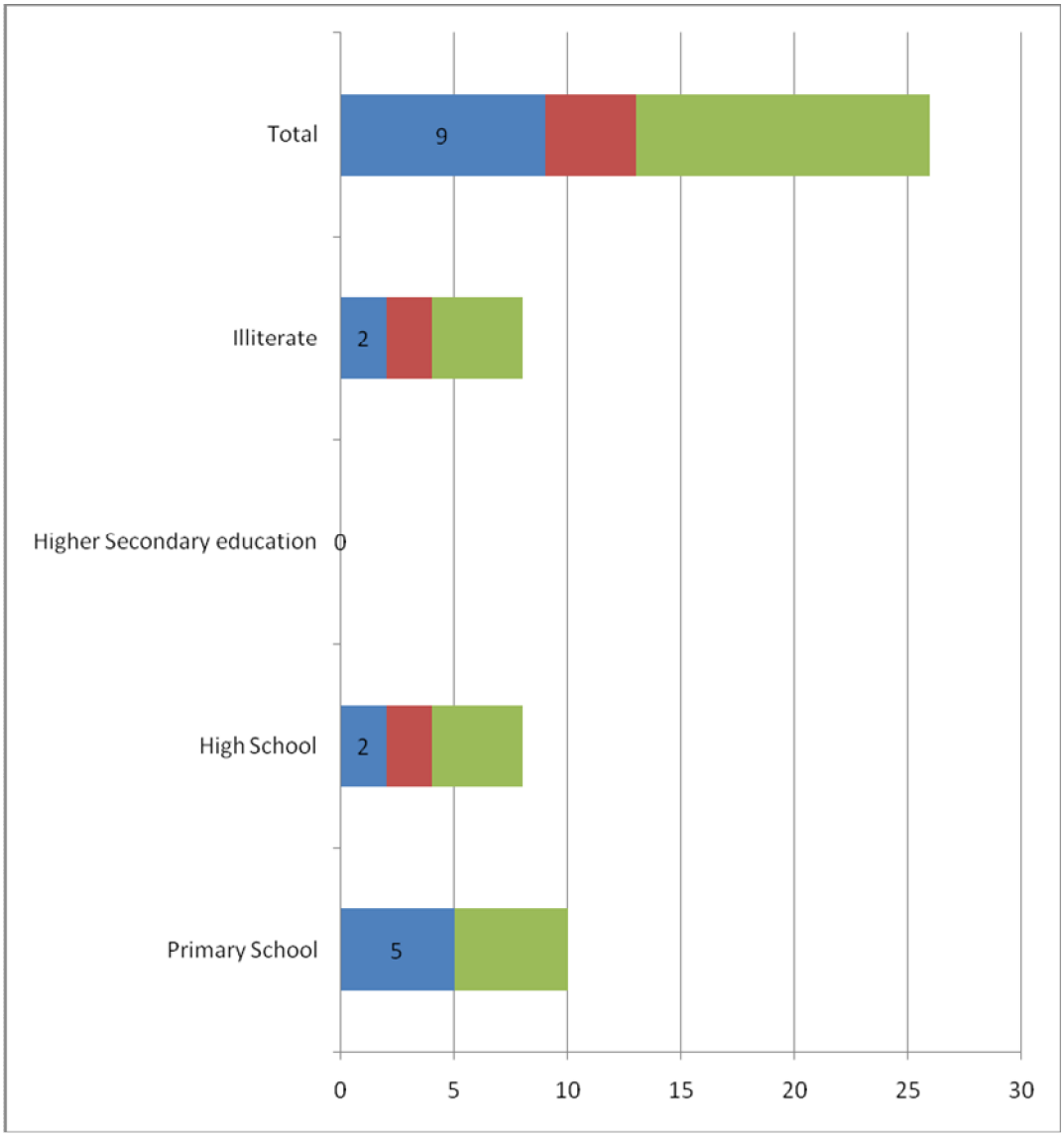
## LITERACY LEVEL IN HBsAg POSITIVE HIV PATIENTS

Out of 13 HBsAg positive HIV patients, 4 were illiterate and the highest level of literacy among others were upto primary school with 5(38.4%) patients followed by high school with 4(30.7%) patients.

**Table 17: LITERACY LEVEL IN HBsAg POSITIVE HIV PATIENTS**

| EDUCATION | MALE | FEMALE | TOTAL |
|---|---|---|---|
| Primary School | 5 | - | 5 |
| High School | 2 | 2 | 4 |
| Higher Secondary | - | - | - |
| Illiterate | 2 | 2 | 4 |
| **TOTAL** | **9** | **4** | **13** |

**Figure 17: LITERACY LEVEL IN HBsAg POSITIVE HIV PATIENTS**



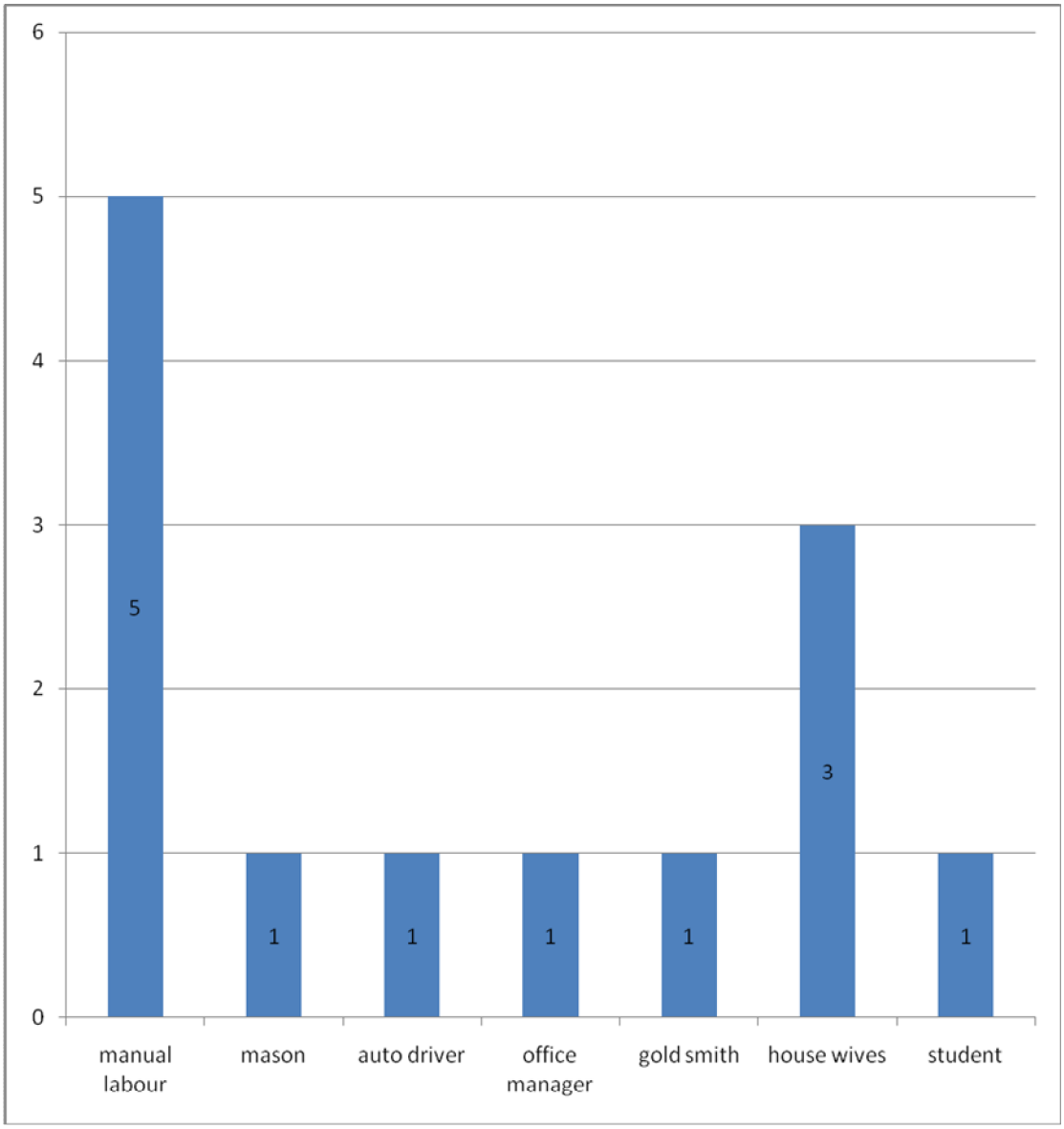
**OCCUPATION IN HBsAg POSITIVE HIV PATIENTS**

Out of 13 HBsAg positive HIV patients, the most common occupation was manual labourer with 5(38.4%) patients.

**Table 18**: **OCCUPATION IN HBsAg POSITIVE HIV PATIENTS**

| OCCUPATION | NO OF PATIENTS |
|---|---|
| Manual labourer | 5 |
| Mason | 1 |
| Auto driver | 1 |
| Office worker | 1 |
| Gold smith | 1 |
| House wives | 3 |
| Student | 1 |
| **TOTAL** | **13** |

**Figure 18 : OCCUPATION IN HBsAg POSITIVE HIV PATIENTS**



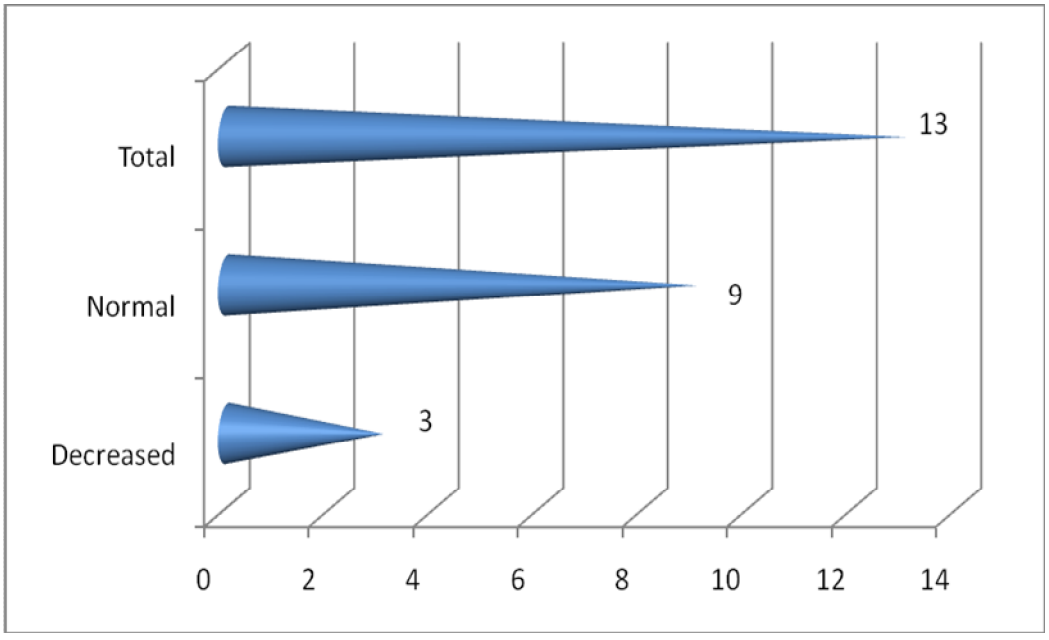
## CD4 COUNT IN HBsAg POSITIVE HIV PATIENTS

Out of 13 HBsAg positive HIV patients, CD4 count was decreased in 3(3%) of patients.

**Table 19: CD4 COUNT IN HBsAg POSITIVE HIV PATIENTS**

| CD4 COUNT | NO OF PATIENTS |
|---|---|
| Decreased | 3 |
| Normal | 96 |
| **TOTAL** | **100** |

**Figure 19: CD4 COUNT IN HBsAg POSITIVE HIV PATIENTS**



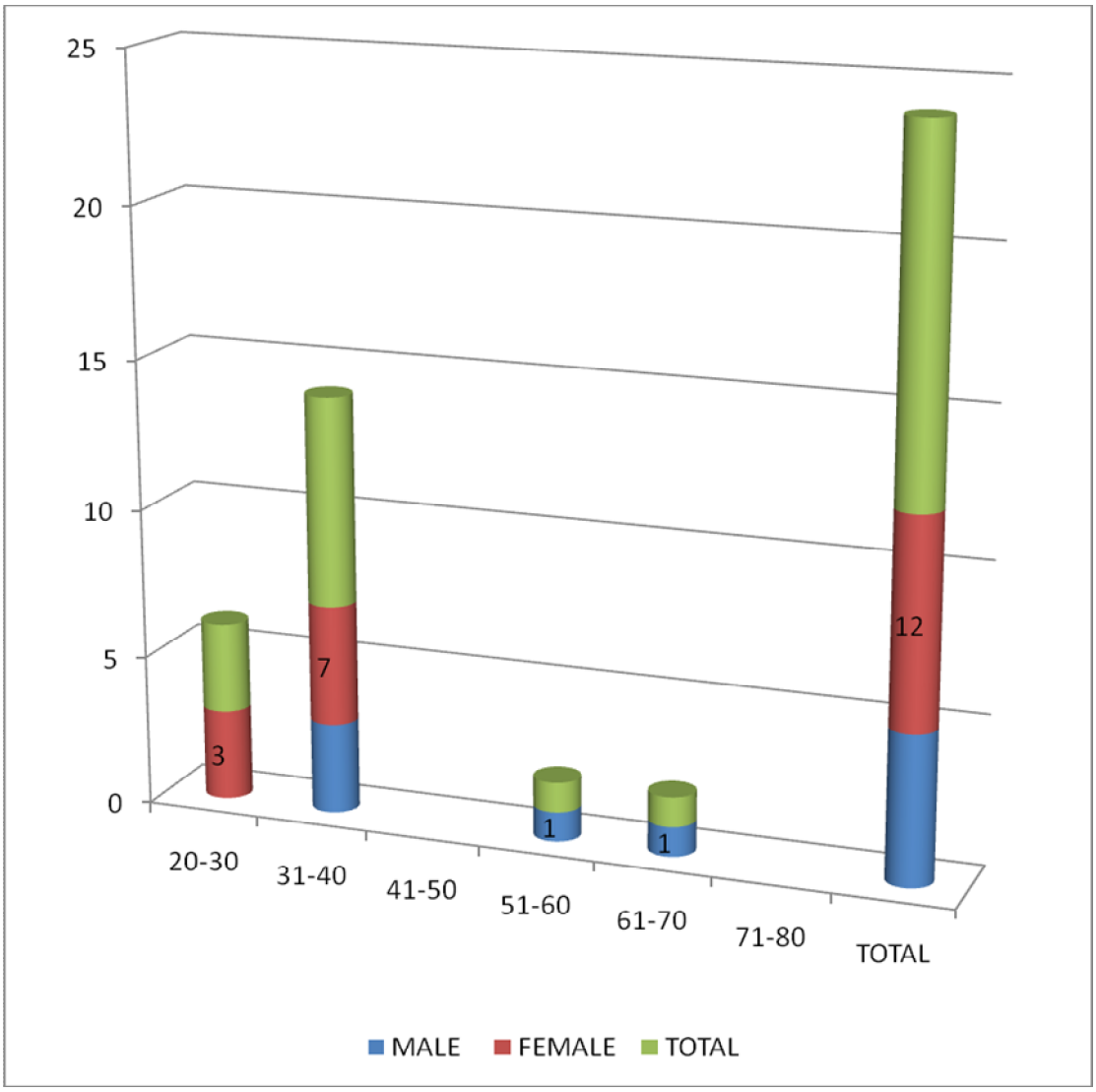
## ASSOCIATED STI CONDITIONS

Associated STI conditions in our study included vulvovaginal candidiasis, bacterial vaginosis, genital wart, genital molluscum contagiosum, candidial balanoposthitis, genital ulcer disease and non specific urethritis. These associated STI conditions were more common in males in the age group of 31 to 40 years with 7(53.8%) patients.

**Table 20: ASSOCIATED STI CONDITIONS**

| AGE | MALE | FEMALE | TOTAL |
|---|---|---|---|
| 20-30 | - | 3 | 3 |
| 31-40 | 3 | 4 | 7 |
| 41-50 | - | - | - |
| 51-60 | 1 | - | 1 |
| 61-70 | 1 | - | 1 |
| 71-80 | - | - | - |
| **TOTAL** | **5** | **7** | **12** |

**Figure 20 : ASSOCIATED STI CONDITIONS**



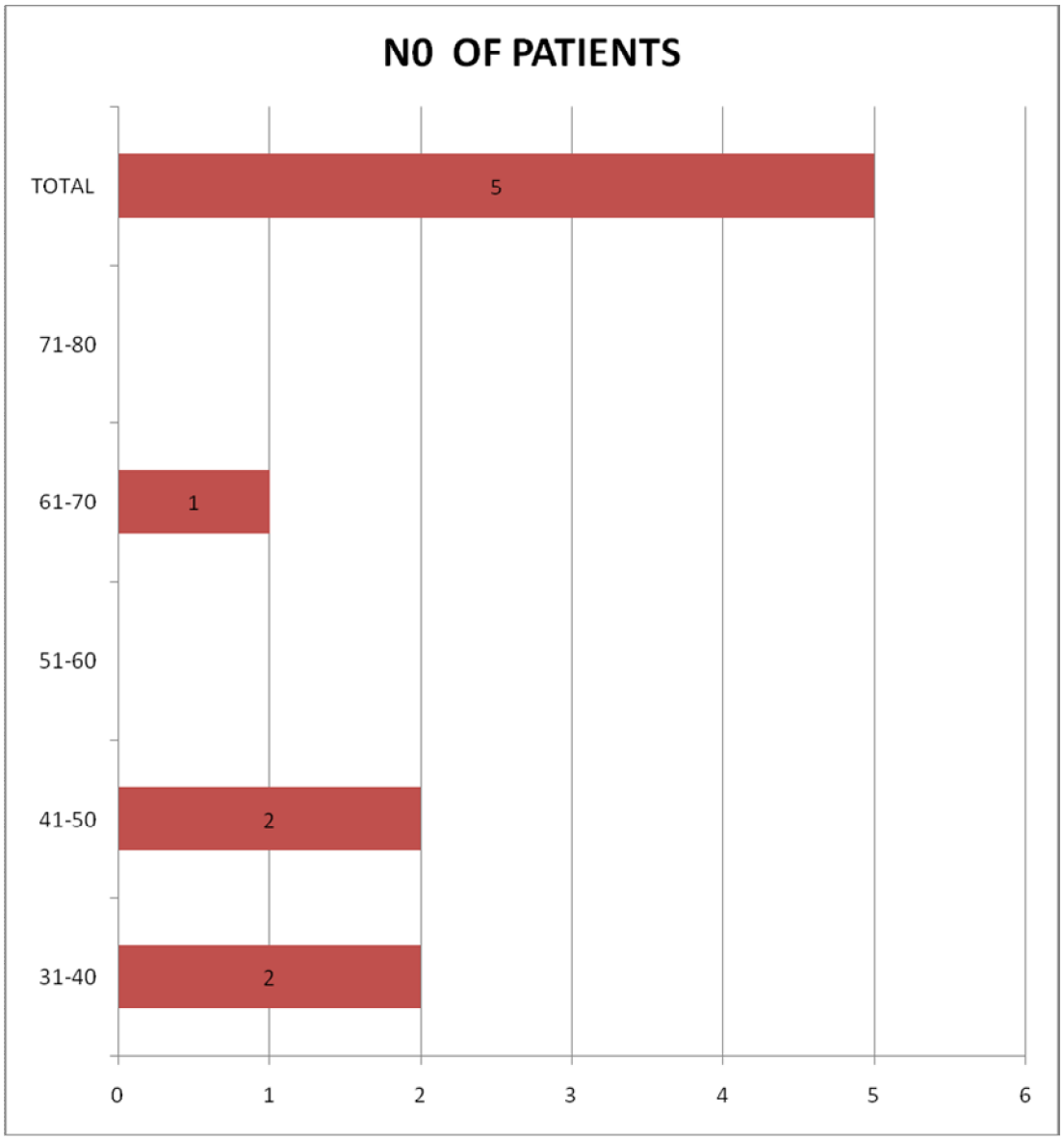
**ASSOCIATED CUTANEOUS FINDINGS IN HIV POSITIVE PATIENTS**

The associated cutaneous conditions in our study included oral candidiasis(2%), tinea cruris(1%), seborrheic dermatitis(1%), herpes zoster(1%), borderline Hansen(1%).

**Table 21: ASSOCIATED CUTANEOUS FINDINGS IN HIV POSITIVE PATIENTS**

| AGE | NO OF PATIENTS |
|---|---|
| 31-40 | 2 |
| 41-50 | 2 |
| 51-60 | - |
| 61-70 | 1 |
| 71-80 | - |
| **TOTAL** | **5** |

**Figure 21: ASSOCIATED CUTANEOUS FINDINGS IN HIV POSITIVE PATIENTS**



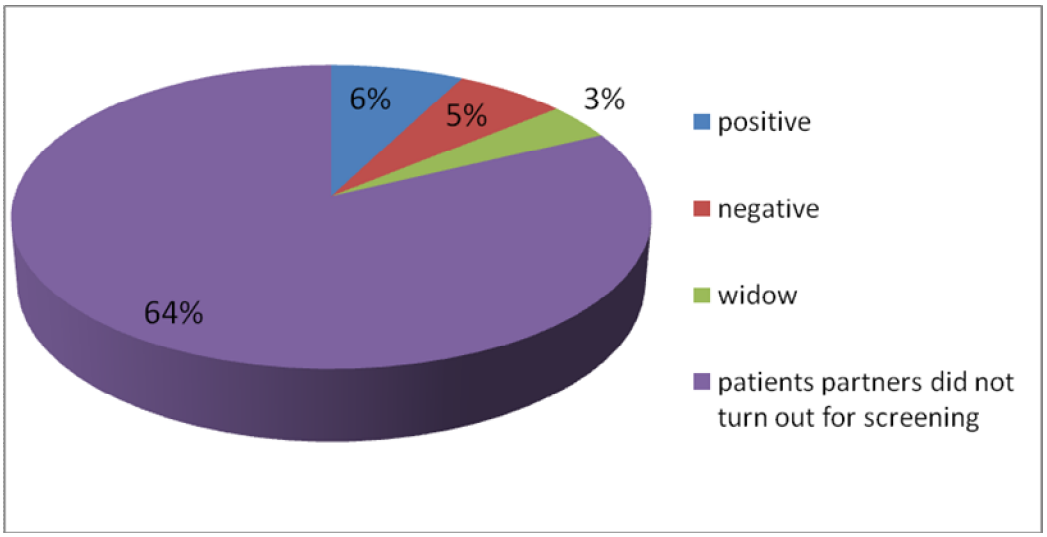
**PARTNER SCREENING IN HIV POSITIVE PATIENTS**

Out of 78 married individuals in our study, 6(6%) patients partners were positive for HIV, 5(5%) patients partners were negative for HIV,3(3%) patients were widow and partners of 64 patients did not turn out for screening inspite of screening advice.

**Table 22 : PARTNER SCREENING IN HIV POSITIVE PATIENTS**

| Partner screening status | Screening Results |
|---|---|
| 6 | positive |
| 5 | negative |
| - | 3 patients were widows |
| 64 | patients partner did not turn out for screening |

**Figure 22: PARTNER SCREENING IN HIV POSITIVE PATIENTS**



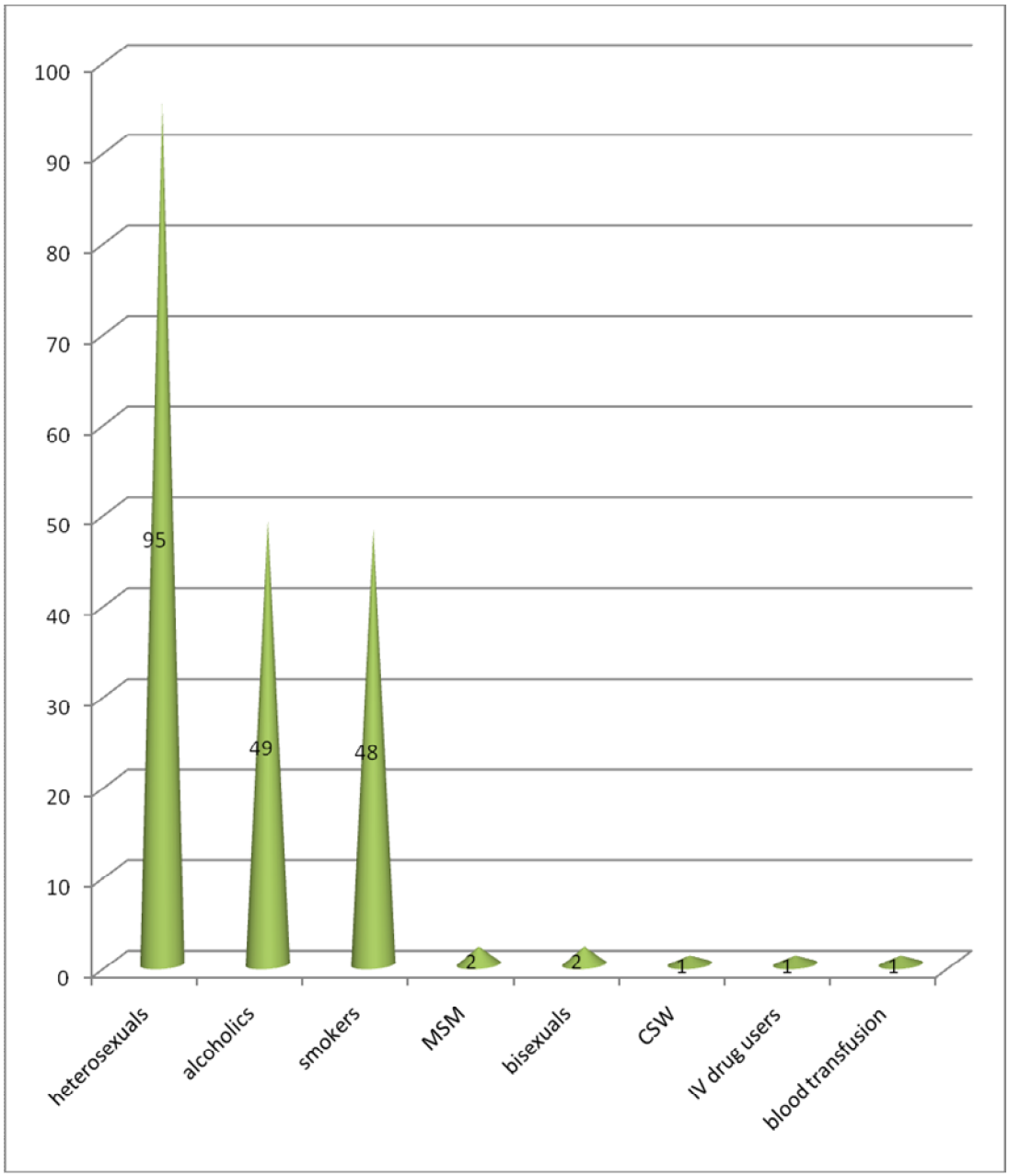
## HIGH RISK GROUPS AMONG HIV POSITVE PATIENTS

Out of 100 HIV infected patients, the major high risk groups were heterosexuals with 95(95%) patients followed by alcoholics 49(49%), smokers 48(48%), MSM 2(2%), bisexuals 2(2%), CSW 1(1%), and patients with blood transfusion1(1%).

**Table 23: HIGH RISK GROUPS AMONG HIV POSITVE PATIENTS**

| HIGH RISK GROUPS | NO OF PATIENTS |
|---|---|
| Heterosexuals | 95 |
| Alcoholics | 49 |
| Smokers | 48 |
| MSM | 2 |
| Bisexuals | 2 |
| CSW | 1 |
| IV drug users | 1 |
| Blood transfusion | 1 |

**Figure 23: HIGH RISK GROUPS AMONG HIV POSITVE PATIENTS**



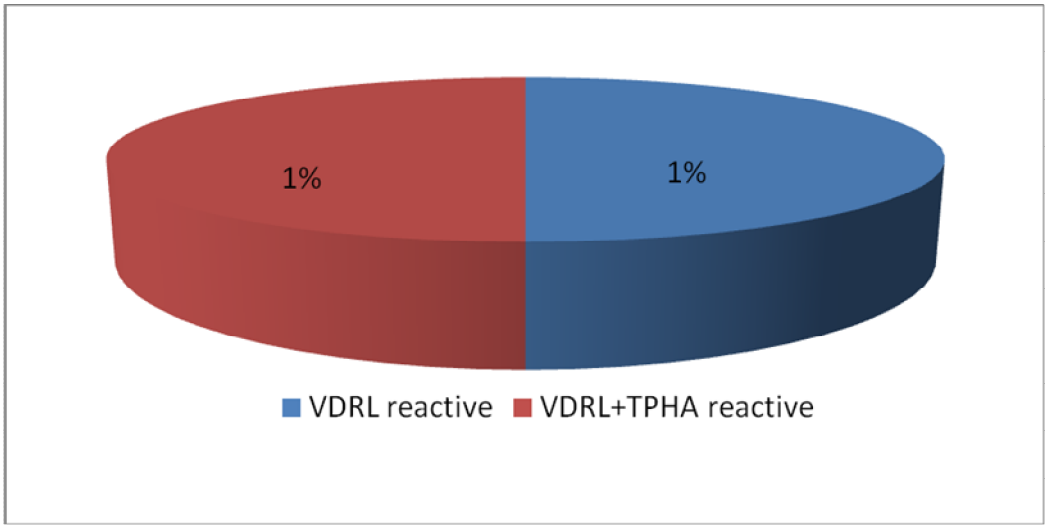
## PREVALENCE OF SYPHILIS IN HIV POSITIVE PATIENTS

Prevalence of Syphilis in HIV positive patients in our study was 2(2%). Out of the two patients, one patient was VDRL reactive (1:16 dilution) and the other patient was both VDRL reactive (1:260 dilution) and TPHA positive.

**Table 24: PREVALENCE OF SYPHILIS IN HIV POSITIVE PATIENTS**

| Test | Result | No of Patients |
|---|---|---|
| VDRL | Reactive | 1 |
| VDRL+TPHA | Reactive | 1 |

**Figure 24: PREVALENCE OF SYPHILIS IN HIV POSITIVE PATIENTS**



# *Discussion*

# DISCUSSION

In this study we included a total of 100 HIV positive patients attending STD clinic. Among these, in HIV positive patients the seroprevalence of Hepatitis B infection was 13(13%).

In a similar study conducted by Sandhya Sawat in Mumbai[63] out of 540 patients, 90(16.7%) patients were positive for Hepatitis B infection.

This is in contrast to a study conducted by Yitayih[64] in Northwest Ethiopia in which the prevalence of Hepatitis B infection was 20(5.6%) patients out of 400 patients.

Whereas a study conducted in Slovenia[65] in 2008 showed a high prevalence of Hepatitis B of about 25.5%[74] and a study by Chiekulie[66] kevin Diwe in South East Nigeria showed a low prevalence of Hepatitis B of about 9(2.2%) patients out of 404 patients.

Thus comparing the above studies a significant high prevalence of Hepatitis B infection was found in our study.

The seroprevalence of hepatitis C in our study out of 100 patients attending STD clinic was 1% in HIV positive patients, which was comparable to a study conducted by Guimaraes[67] Nebenzal in which the

seroprevalence of Hepatitis C infection in HIV positive patients was 4(0.9%) patients out of 431 patients.

In another study conducted by Sandhya Sawat and Sachee Agrawal[63] the concomitant infection of Hepatitis C with HIV was 7(1.3%) out of 540 patients which is very identical to our study.

Whereas a study conducted by Seme K, Lunar MM Slovenia[65] reported a high prevalence in comparison to our study of 10.7% which can be attributed to the lack of effective vaccine and other treatment modalities against Hepatitis C infection. In a study done by Yitayih[64] in the year 2011 which was conducted in a study population of about 400 individuals reported 5% co-infection of Hepatitis C with HIV.

Thus the wide range of prevalence in Hepatitis B and Hepatitis C infection can be due to variation in individuals immunity, exposure to risk factor, variation in the geographic region, associated underlying disease and also due to different routes of exposure to infection.[75-78]

The combined infection of Hepatitis B and Hepatitis C was 1% in our study which was similar to a study of 0.5% (1 out of 200 patients) conducted by Sumit Goyat[68] and 0.9%(4 out of 431 patients) conducted by Guimaraes.[67]

Triple infection HIV, Hepatitis B, and Hepatitis C in our study was 1% which was similar to a study done by Guimaraes[67] which was conducted in the age group between 15 to 49 years in which 5 of them out of 431 i.e 1.2% patients showed triple infection of HIV, Hepatitis B, and Hepatitis C. Another study done by Sandhya Sawat[63] showed the concomitant infection of HIV, Hepatitis B, and Hepatitis C as 0.4% (2 out of 540 patients) respectively. But a study done by Chiekulie[66] kevin Diwe in South East Nigeria reported that no participants in his study had triple infection of HIV, Hepatitis B, and Hepatitis C.

In our study the prevalence of Syphilis in HIV positive patient was 2(2%) which was comparable to a study conducted by Guimaraes[67] in which 2 individuals had Syphilis and HIV co-infection out of 431 patients (0.4%).

In another study conducted by Nancy crum-cianflone[69] the prevalence of Syphilis in HIV positive patient was 6% (33 out of 600 patients) which was relatively closer to the studies conducted by Alemayehu A, Shimelis T[85] and Bjekić M, Marković M[86] which reported a prevalence of 9.8% and 15.3% respectively.

In contrast to these studies a study conducted by D.T urbadkar[70] showed a high prevalence of 47% (42 patients out of 88 HIV seropositive cases).

Thus compared to the above studies a low prevalence of Syphilis in HIV positive patient was noticed in our study which is due to the effective antibiotic era at present.

In our study Liver enzyme level was elevated in 4(4%) HIV positive patients and 3(3%) of HIV-HbSAg positive patients.

Among these 4(4%) HIV positive patients 3 patients showed elevated SGPT (with 60 IU, 76 IU, and 120 IU), SGOT (with 80 IU, 68 IU and 94 IU) and alkaline phosphatase(with 133,140 and 166 IU) and 1 patient showed isolated elevation of alkaline phosphatase(140 IU) with normal levels of SGPT and SGOT

Among the HIV-HbSAg positive patients all 3 patients showed elevated SGPT (with 60 IU, 76 IU, and 120 IU), SGOT (with 80 IU, 68 IU and 94 IU) and alkaline phosphatase (with 133,140 and 166 IU) levels.

Out of the 3 liver enzymes there are literature reporting that alkaline phosphatase is most important and commonly associated liver enzyme in HIV positive patients[104] .Where as SGPT is more commonly

associated with alcoholic liver disease. Similarly in a study conducted by Ranjit pateel showed that alkaline phosphatase was elevated in 35% above the normal limit in HIV positive patient and in another study conducted by Payne T H,[72] showed a marked elevation of serum ALP in excess of 1000IU/L in 17% of AIDS patient.

## DEMOGRAPHIC CHARACTERS

### AGE

The most common age group involved in our study in HIV infected individuals were 31-40 years which was similar to a study conducted by Yitayih[64] were the study participants were in the age group of 30-39years while 25- 35 years age group participants were common in a study conducted by Sandhya Sawat. [63]

### SEX

Out of 100 patients HIV was more common among males with 70(70%) patients while females were 30(30%) patients which was very much similar to a study conducted by Saravanan s Velu [73]and Sumit Goyat[68] with male preponderance of 73% and 65.5% but a study conducted by Chiekulie[66] kevin Diwe showed a female preponderance with female 69.2% and male 30.8% patients respectively.

## OCCUPATION

The study participants in our study were most of them who belonged to manual labourer occupation with 40 patients belonging to manual labour, followed by mason with 7 patients , auto driver with 5 patients, electricians with 4 patients, truck driver with 3 patients and farmer with 2 patients and others belonged to mechanic, painter, hotel worker, gold smith, accountant and artist, apart from these there were 5 students and 16 house wives, whereas a study conducted by Amar Surjushe[74] among the 40 male patients, 20 were manual laborers and 14 were farmers, and among the 20 female cases, 18 patients were housewives. In another study conducted by Chiekulie[66] kevin Diwe out of 404 patients commercial drivers constituted about 7.6%, public servants 12.6%, students were about 10.5% and 69% belonged to farmers, traders and artisans.

## LITERACY

The highest level of formal education attained by our study participants were high school level with 33(33%) patients which was next followed by primary school level with 26(26%) and illiterate constituted about 34(34%) patients.

In a study conducted by Chiekulie[66] kevin Diwe there was a close similarity to our study with primary education level to 27.3%, secondary

education upto 55.3% and 3.6% constituted illiterate and a study conducted by Yitayih[64] showed HIV infected individuals with higher secondary education and diploma constituted to about 4(10.3%) patients .

Another study reported by Goyal Ankur and Goyal Sapna[79] in a study group of 358 HIV infected patients showed that 89.1% were illiterate and below 8 $^{th}$ standard, whereas a study in Brazil[80] reported that 74% of the HIV cases educational level were illiterate or had hardly completed middle school.

Thus in our study the percentage of illiterate was slightly higher than educated individuals which stresses the need to create awareness about the importance of education among these individuals since lack of education plays a vital role in the occurrence of STIs .

**HIGH RISK GROUPS**

**MSM**

In our study a low prevalence of MSM of about 2(2.8%) was noted out of 70 HIV infected male patients and no MSM were noticed in Hepatitis B and Hepatitis C infected patients, and MSM in Syphilis was seen only in one patient.

In a study conducted by Sumit Goyat[68] the prevalence of MSM in HIV infected patients was 1% and the prevalence of MSM in Hepatitis B infected patients was 1(11.11%) out of 9 patients and none of Hepatitis C infected patients was MSM.

Whereas a study conducted by Saje A, Tomazic[81] J in Syphilis patients showed relatively high prevalence of MSM of about 76.3%, another study conducted by Taru Garg**,** Ram and Chander**,** Arpita Jain[82] showed prevalence as 75(11.4%) patients out of 660 patients.

**HETEROSEXUALS**

In our study majority of HIV infected patients were heterosexuals with 95(95%) patients and all the 13 Hepatitis B infected patients and one Hepatitis C infected patient were heterosexuals. In a study done by Sandhya Sawat[63] showed that heterosexuals constituted 80.6% out of 540 patients, whereas a study conducted by Sumit Goyat[68] showed the heterosexuals rates as 72%, 66.7% and 53.3% in HIV, Hepatitis B, and Hepatitis C infected patients respectively.

**BISEXUALS**

The prevalence of bisexuals in our study was 2(2%) patients in HIV infected individuals and none of these two patients had Hepatitis B and Hepatitis C infection or Syphilis. Similarly in a study conducted by

Raj Narayan, and Deepak Mathur[83] the prevalence of bisexuals was 2(2%) out of 100 patients. In another study conducted by Sumit Goyat[68] prevalence of bisexuals was 1% among HIV infected patients.

**COMMERCIAL SEX WORKERS (CSW)**

The prevalence of commercial sex workers(CSW) in our study was 1(1%) among HIV infected patients but this patient did not have any co-infection with Hepatitis B, Hepatitis C or Syphilis, whereas in a study conducted by Raj Narayan, and Deepak Mathur[83] reported the prevalence of commercial sex workers(CSW) to be 28% in HIV infected patients and 40% in VDRL reactive patients, similarly in a study conducted by Paritosh Kumar Banerjee, and Manol Kumar Mandal[84] the prevalence of commercial sex workers(CSW) was 3% in Calcutta and a study conducted in Mumbai reported 18% prevalence of commercial sex workers(CSW).

Though a low prevalence of 1(1%) commercial sex workers (CSW) was reported in our study compared to a significantly high prevalence of 18% in Mumbai but since commercial sex workers (CSW) form a vital reservoir of many STIs their effective screening and timely intervention of treatment is important.

## TRANSGENDERS

There was no history suggesting the occurrence of HIV seropositivity among Transgenders in our study however a study conducted by Carobene M, Bolcic F, Farías MS[89] among sex workers in Argentina showed the prevalence of Transgenders in Hepatitis B with HIV co-infection as 3.2% and Hepatitis C with HIV co-infection as 6.5% respectively. In another study conducted by Fabiana Schuelter-Trevisol[90] in Brazil among 147 patients showed prevalence of Transgenders as 7(4.3%) patients.

## INTRAVENOUS DRUG USERS (I.V drug users)

In our study a single patient gave history of intravenous drug abuse who had HIV seropositivity but had no co-infection of Hepatitis B, Hepatitis C or Syphilis which was comparable to the study conducted by Sandhya Sawat[1] which showed a prevalence of about 2(0.4%) out of 540 patients.

In a study conducted by Sumit Goyat[68] showed a prevalence of 2.5%, 22.2% and 20% in HIV, Hepatitis B, and Hepatitis C infected patients whereas a study done by Raj Narayan, and Deepak Mathur[83] which was conducted in 100 patients had no patients with I.V drug users.

## BLOOD TRANSFUSION

In our study only one patient out of 100 patients i.e 1% patient gave history of blood transfusion who had HIV seropositivity but had no co-infection of Hepatitis B, Hepatitis C or Syphilis which to some extent is similar to the study conducted by Sandhya Sawat[63] where among a study group of 540 HIV infected patients 15(2.8%) patients had history of blood transfusion. In another study done by Sen et al[87] showed about 8.9% patients had received blood transfusion who were infected with HIV. Although prevalence of HIV infected patients who received blood transfusion is low in our study, there are study based evidence suggesting that through a single unit of blood transfusion there is about 1% chance of getting STI related problems,[88] Hence prompt screening of blood donors are emphasized in order to decrease the occurrence blood transfusion related STIs.

## PERINATAL TRNSMISSION

Perinatal transmission is an important route for transmission of HIV other STIs. In our study though there was no history of perinatal transmission among the 25 married women. Similar to our study, a study conducted by Sandhya Sawat[63] out of 540 HIV infected patients proved no history of perinatal transmission however a study conducted by Sumit

Goyat[68] showed out of 200 patients, 4% women gave history of vertical transmission in HIV infected patients.

**ALCOHOLICS**

Among the 70 male patients in our study 49(70%) patients were alcoholics with HIV seropositivity and the maximum number of patients belonged to 20-30 years with 15(21.4%) patients, this was comparable to a study conducted by Fabiana Schuelter-Trevisol[90] in Brazil in which 83.7% patients among 147 patients were alcoholics.

Out of 13 HBsAg positive patients 8(61.5%) patients were alcoholics with maximum no of patients in the age group of 41-50 years with 2(25%) patients and only one patient had anti-HCV antibodies who was an alcoholic.

Thus the prevalence of alcoholics in our study among HIV, Hepatitis B, and Hepatitis C infected patients were 70%, 61.5% and 1% respectively and none of the VDRL reactive patients were alcoholics. This significant percentage of alcoholics is thus an important risk factor for STIs and associated with liver damage on chronic use.

**SMOKERS**

Among the 70 male patients in our study 48(68.5%) patients were smokers with HIV seropositivity and the most commonly affected age

groups were 31-40years with 13(18.5%) patients and out of 13 HBsAg positive patients 8(61.5%) patients were smokers and the maximum no of patients belonged to $2^{nd}$, $4^{th}$ and $5^{th}$ decade with 2(25%)patients and none of the patients had anti-HCV antibodies or VDRL reactivity. Though smoking is not directly related to STIs chronic smoking is associated with various pulmonary complication and decreased immunity which in turn contributes to increased chance for the occurrence of STIs.

**CD4 COUNT**

In our study out of 13 HIV-HBV infected patients, 3 patients showed markedly decreased value of CD4 count (of 191, 160, 170cells/$mm^3$) and all these three patients were also chronic alcoholics which further contributes to liver damage. The mean CD4 count among these 13 patients was 268 which was very similar to a study conducted by Yitayih[64] in which the mean CD4 count among 400 patients was 250 in contrast to a study conducted by Lodenyo H, Schoub B, Ally R, Kairu S[91], and Olufemi A, Emmanuel A[92] in which the mean CD4 count was 141.6 and 121cells/$mm^{3.}$ The mean CD4 count in our study in HIV-HCV infected patients was 170cells/$mm.^3$

Whereas a study conducted by Olufemi A, Emmanuel A[92] and Tripathi A, Khanna M[93] reported a mean CD4 count of 260 and 288.6cells/$mm^3$ respectively. The mean CD4 count in our study were

relatively lower in males when compared to females (258vs291) similar difference in gender were noticed in the studies conducted by Akinsegun, Adedoyin D[94] and Tugume SB[95] respectively.

## ASSOCIATED STI CONDITIONS

The occurrence of HIV, Hepatitis B, and Hepatitis C and Syphilis increases the susceptibility of getting other STIs like in our study 12 patients presented with a number of other viral, fungal and bacterial STIs like vulvovaginal candidiasis (4%), bacterial vaginosis (3%), genital wart1(1%), genital molluscum contagiosum 1(1%), candidial balanoposthitis1(1%), genital ulcer disease 1(1%) and non specific urethritis 1(1%) .

In a study done by Raj Narayan, and Deepak Mathur[83] which was conducted in 100 patients showed associated conditions like 23(23%) cases of genital warts, 2(2%) cases of Non specific urethritis 22(22%)cases of chancroid,15(15%)cases of genital herpes, 10(10%) cases of balanoposthitis and 8(8%) cases of gonorrhea.

## ASSOCIATED CUTANEOUS CONDITIONS

Common dermatological conditions like oral candidiasis(2%), tinea cruris (1%), seborrheic dermatitis(1%), herpes zoster(1%), borderline Hansen(1%) were seen among 5 out of 100 patients, among these oral

candidiasis was the commonest cutaneous finding which was also a commoner cutaneous condition in a number of Indian studies.[96,97,98,99]

Similarly another study conducted by Shobhana A, Guha SK[100] showed various cutaneous conditions in HIV infected patients like oral candidiasis, dermatophytosis and gingivitis (13% each), herpes zoster (6%), herpes simplex, scabies (5% each), molluscum contagiosum, staphylococcal skin infections, oral herpetic lesions, Papular pruritic dermatoses and guttate psoriasis. A study conducted by Kar PK, Ramasastry CV[101] showed a high prevalence of herpes zoster of 11(9.5%) out of 115 patients when compared to 1% in our study. Similarly tinea cruris was present in only 1% of our case compared to a very high prevalence of about 56.66% conducted by Amar Surjushe.[102]

# *Summary*

# SUMMARY

- In this study out of 100 HIV positive patients the prevalence of Hepatitis B was 13%, Hepatitis C was 1%, Syphilis was 2% and elevated liver enzymes was reported in 4(4%) patients.

## DEMOGRAPHIC CHARACTERS

### AGE GROUP

- The most common age group among HIV positive patients was 3rd decade with 33%.
- The most common age group among HBV positive HIV patients was $4^{th}$ decade with 38.6%.
- The most common age group among HCV positive HIV patients was 4th decade with 1% .
- The most common age group among VDRL reactive patients was 3rd decade with 2%.

### SEX

- In our study males were predominantly affected than females.
- 70% were males and 30% were females in HIV positive patients.
- 69.2% males and 30.7% females in HBV positive HIV patients.

- 2% males in Syphilis and 1% males in HCV positive HIV patients were reported with no females affected.

**OCCUPATION**

- The most common occupation among HIV positive patients was manual labour (31%) in males followed by masons (7%), auto drivers (5%) and truck drivers(3%) and females were predominantly house wives (16%) followed by manual labourers (9%).
- The predominant occupation among HBV positive HIV patients was manual labour (5%) in males and females (4%).
- Similarly manual labourers was the predominant occupation in HCV positive and VDRL reactive patients with 1% and 2% respectively.

**LITERACY**

- The highest education level among HIV positive patients was upto high school with 33( 33%) patients.
- The highest education level among HBV positive HIV patients was upto primary school with 5(38.4%) patients.
- The highest education level among HCV positive HIV patients was upto primary school with 1(1%) patients.
- The highest education level among VDRL reactive patients were upto primary school with 1(1%) patients.

## HIGH RISK GROUPS

### a) MSM

- Prevalence of MSM was 2(2.8%) patients out of 70 HIV positive male patients.
- No MSM was noticed in Hepatitis B and Hepatitis C infected patients.
- MSM in Syphilis was seen only in one patient.

### b) HETEROSEXUALS

- In our study prevalence of heterosexuals in HIV positive patients was 95(95%) patients.
- Prevalence of heterosexuals in HBV and HCV positive HIV patients was 13(100%) patients and 1(100%) patients respectively.
- Prevalence of heterosexuals in VDRL reactive HIV patients was 1(50%) patients.

### c) BISEXUALS

- The prevalence of bisexuals among HIV positive patients was 2(2%) patients .
- None of the HBV, HCV positive HIV patients and VDRL HIV reactive patients were bisexuals.

**d) COMMERCIAL SEX WORKERS(CSW)**

- The prevalence of commercial sex workers(CSW) in our study was 1(1%) among HIV infected patients.
- No commercial sex workers (CSW) were reported among the HBV, HCV positive and VDRL reactive HIV patients.

**e) INTRAVENOUS DRUG USERS(I.V drug users)**

- The prevalence of I.V drug users among HIV positive patients was 1(1%).
- None of the HBV, HCV positive HIV patients or VDRL reactive HIV cases were reported among I.V drug users.

**f) BLOOD TRANSFUSION**

- 1(1%) patient gave history of blood transfusion among HIV positive patients with no case reported among HBV, HCV positive HIV patients or VDRL reactive HIV patients.

**g) ALCOHOLICS**

- Prevalence of alcoholics in our study among HIV, HBV, and HCV positive patients were 70%, 61.5% and 1% respectively and none of the VDRL reactive HIV patients were alcoholics.

**h) SMOKERS**

- Prevalence of smokers were 48(68.5%) patients in HIV positive patients and 8(61.5%) patients in HBV positive HIV patients no smokers were reported in HCV positive HIV patients or VDRL reactive HIV patients.

**i) ASSOCIATED STI CONDITIONS**

- The associated STI conditions in our study included vulvovaginal candidiasis (4%), bacterial vaginosis(3%), genital wart 1(1%), genital molluscum contagiosum 1(1%), candidial balanoposthitis1(1%), genital ulcer disease 1(1%) and non specific urethritis 1(1%) .

**j) ASSOCIATED CUTANEOUS CONDITIONS**

- The associated cutaneous conditions in our study included oral candidiasis(2%), tinea cruris(1%), seborrheic dermatitis(1%), herpes zoster(1%), borderline Hansen(1%).

**k) CD4 COUNT**

- CD4 count was decreased among 3(23%) HBV positive HIV patients and 1(1%) HCV positive HIV patients.

# *Conclusion*

# CONCLUSION

- In our study group of 100 HIV positive patients, the prevalence of Hepatitis B was 13%, Hepatitis C was 1%, Syphilis was 2% and elevated Liver Enzymes was 4% .
- Most common affected age group was 3$^{rd}$ and 4$^{th}$ decade.
- Males were predominantly affected than females.
- Most common occupation among males was manual labour, whereas females were predominantly house wives.
- The highest level of education was upto high school and primary school and the literacy level among males was comparatively higher than females.
- The major High Risk Group was heterosexuals followed by alcoholics, smokers, MSM, bisexuals , CSW, and patients with blood transfusion.
- Associated STI conditions included vulvovaginal candidiasis, bacterial vaginosis, genital wart, genital molluscum contagiosum, candidial balanoposthitis, genital ulcer disease and non specific urethritis.
- Associated cutaneous conditions included oral candidiasis, tinea cruris, seborrheic dermatitis, herpes zoster, and borderline Hansen.
- CD4 count was markedly decreased in 3 patients.

- Thus to conclude in our study,

1) HIV-HBV co-infection was seen in 13(13%) patients.
2) HIV-HCV co-infection was seen in 1(1%) patient.
3) HIV with Syphilis co-infection was seen in 2(2%) patients.
4) Elevated liver enzyme levels in HIV positive patients were seen in 4(4%) patients.
5) Decreased CD4 count was seen seen in 3(3%) patients.
6) HIV-HBV co-infection with elevated liver enzyme levels and decreased CD4 count was seen in 3(3%) patients.
7) HIV-HCV co-infection with elevated liver enzyme levels and decreased CD4 count was seen in 1(1%) patient.
8) HIV-HBV-HCV co-infection with elevated liver enzyme levels and decreased CD4 count was seen in 1(1%) patient.

The decreased CD4 count with elevated liver enzyme levels as seen in our study will cause further liver damage, hence ART should be immediately started in these patients. However according to WHO guidelines[41] patients with combined HIV-HBV or HIV-HCV infection should be started on ART irrespective of the CD4 count.

Hence routine screening for Hepatitis B and Hepatitis C should be emphasized in HIV positive patients since its early detection can decrease the morbidity and mortality due to liver damage among these patients.

Routine screening for Syphilis should also be encouraged in HIV positive patients since its early detection can decrease the morbidity and mortality among these patients.

By routine screening effective treatment can be implemented to increase the life span and quality of life among such patients .

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# *Annexures*

# ABBREVIATIONS USED

| | | | |
|---|---|---|---|
| 1. | HIV | : | Human Immunodeficiency Virus |
| 2. | HART | : | Highly active Antiretroviral Theraphy |
| 3. | MSM | : | Men having Sex with Men |
| 4. | VDRL | : | Venereal Disease Research Laboratory |
| 5. | HBV | : | Hepatitis B Virus |
| 6. | HCV | : | Hepatitis C Virus |
| 7. | HBsAg | : | Hepatitis B surface antigen |
| 8. | HBeAg | : | Hepatitis B envelope antigen |
| 9. | Anti-HBsAb | : | Hepatitis B surface antibody |
| 10. | Anti-HBcAb | : | Hepatitis B core antibody |
| 11. | PCR | : | Polymerase Chain Reaction |

# MASTER CHART

| SL.NO | NAME | AGE | SEX | MARITAL STATUS | EDUCATION | OCCUPATION | HRG | PARTNER SCREENING | HIV+HBV POSITIVE | HIV+HCV POSITIVE | HIV+VDRL POSITIVE | LIVER ENZYME LEVELS | | | STI SYMPTOMS | CUTANEOUS FINDING | VDRL |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | | | | | SGOT | SGPT | ALP | | | |
| 1 | Senthil | 25 | male | UM | 10th | mason | A+S | - | - | - | - | 17 | 10 | 75 | - | -- | NR |
| 2 | Karthik | 27 | male | M | 10th | electrician | A | - | - | - | - | 68 | 27 | 75 | - | - | NR |
| 3 | Suraj | 45 | male | M | 10th | clerk | A | - | - | - | - | 14 | 12 | 70 | - | - | NR |
| 4 | Prabhu | 25 | male | M | 8th | manual labourer | S | - | - | - | - | 16 | 24 | 72 | - | - | NR |
| 5 | Deva | 38 | male | M | - | mechanic | A+S | - | - | - | - | 27 | 24 | 60 | - | | NR |
| 6 | Allimuthu | 30 | male | M | 7th | manual labourer | A+S | - | positive | - | - | 18 | 20 | 41 | - | BL leprosy | NR |
| 7 | Surrulivel | 60 | male | M | 4th | manual labourer | A+S | - | - | - | - | 28 | 26 | 47 | - | - | NR |
| 8 | Selvaraj | 63 | male | M | - | manual labourer | A+S | - | positive | - | - | 60 | 80 | 92 | - | candidiasis | NR |
| 9 | Yesudev | 38 | male | M | - | manual labourer | A+S | negative | - | - | - | 28 | 18 | 64 | - | | NR |
| 10 | Veeran | 33 | male | M | 10th | accountant | A+S+IVD | - | - | - | - | 30 | 34 | 64 | - | candidiasis | NR |
| 11 | Ganesh | 28 | male | M | - | mason | A+S | - | - | - | - | 76 | 68 | 133 | - | - | NR |
| 12 | Raju | 75 | male | UM | - | mason | A+S | - | - | - | - | 23 | 14 | 41 | - | - | NR |
| 13 | Babu | 39 | male | M | 6th | manual labourer | A+S | - | - | - | - | 14 | 68 | 47 | - | HZ | NR |
| 14 | Murugan | 50 | male | M | 7th | manual labourer | A+S | - | positive | - | - | 24 | 26 | 40 | - | - | NR |
| 15 | Thirupathy | 32 | male | M | 6th | mason | - | - | - | - | - | 14 | 17 | 47 | - | - | NR |
| 16 | Gopi | 44 | male | M | 8th | manual labourer | S | - | - | - | - | 17 | 13 | 43 | - | - | NR |
| 17 | Boobalan | 47 | male | M | 10th | auto driver | - | - | - | - | - | 18 | 12 | 49 | - | - | NR |
| 18 | Mani | 63 | male | M | -- | manual labourer | S | - | - | - | - | 34 | 24 | 41 | - | - | NR |
| 19 | Pandiyan | 35 | male | M | 2nd | electrician | S | - | - | - | - | 24 | 38 | 42 | - | - | NR |
| 20 | Jagan | 24 | male | M | 10th | auto driver | S | - | - | -- | - | 16 | 20 | 33 | - | - | NR |
| 21 | Susendran | 42 | male | M | 10th | farmer | S | - | positive | - | - | 19 | 20 | 26 | - | - | NR |
| 22 | Senthil | 28 | male | M | 5th | mason | S | - | - | - | - | 15 | 26 | 30 | - | - | NR |
| 23 | Sivakumar | 39 | male | M | 8th | auto driver | A | - | - | - | - | 19 | 20 | 26 | genital wart | - | NR |
| 24 | Bakiyaraj | 34 | male | UM | - | manual labourer | - | - | - | - | - | 14 | 17 | 47 | - | - | NR |

| SL.NO | NAME | AGE | SEX | MARITAL STATUS | EDUCATION | OCCUPATION | HRG | PARTNER SCREENING | HIV+HBV POSITIVE | HIV+HCV POSITIVE | HIV+VDRL POSITIVE | LIVER ENZYME LEVELS | | | STI SYMPTOMS | CUTANEOUS FINDING | VDRL |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | | | | | SGOT | SGPT | ALP | | | |
| 25 | Venkateshan | 52 | male | M | - | manual labourer | MBT | - | - | - | - | 25 | 26 | 30 | - | - | NR |
| 26 | Mohan | 53 | male | M | 8th | office worker | - | - | positive | - | - | 51 | 64 | 140 | candidial balanopost hitis | - | NR |
| 27 | Ezhil | 45 | male | M | 10th | manual labourer | S | - | - | - | - | 33 | 24 | 30 | - | - | NR |
| 28 | Subbaiah | 55 | male | UM | - | manual labourer | A+S | - | - | - | - | 26 | 16 | 46 | - | - | NR |
| 29 | Marimuthu | 48 | male | M | 12th | mason | A+S | - | - | - | - | 19 | 33 | 38 | - | - | NR |
| 30 | Vasudevan | 47 | male | M | 8th | electrician | A | - | - | - | - | 22 | 16 | 80 | - | - | NR |
| 31 | Murali | 39 | male | M | 7th | manual labourer | A+S | - | positive | - | - | 120 | 94 | 140 | - | - | NR |
| 32 | Raja | 31 | male | M | 11th | electrician | A+S | - | - | - | - | 24 | 34 | 46 | - | - | NR |
| 33 | Michel | 26 | male | M | 12th | manual labourer | A+S | - | - | - | - | 16 | 20 | 39 | - | - | NR |
| 34 | Chakravathy | 29 | male | M | 10th | auto driver | A+S | - | - | - | - | 10 | 20 | 39 | - | T.cruris+SD | NR |
| 35 | Mani | 24 | male | M | 8th | auto driver | A+S | - | positive | - | - | 50 | 21 | 64 | - | - | NR |
| 36 | Harinathan | 32 | male | M | - | manual labourer | - | - | - | - | - | 26 | 20 | 41 | MC left scrotum | - | NR |
| 37 | Saravanan | 44 | male | M | 7th | manual labourer | A+S | - | - | - | - | 21 | 17 | 20 | - | - | NR |
| 38 | Poncilla | 69 | male | M | 10th | security | A+S | positive | - | - | - | 18 | 13 | 39 | - | - | NR |
| 39 | Gnanaraj | 31 | male | M | 8th | security | A | - | - | - | - | 17 | 39 | 48 | - | - | NR |
| 40 | Vadivel | 37 | male | M | 6th | manual labourer | A+S | - | - | - | - | 18 | 20 | 41 | - | - | NR |
| 41 | Muthu | 48 | male | M | - | farmer | A+S | - | - | - | - | 21 | 28 | 60 | - | - | NR |
| 42 | Balwir | 53 | male | M | 9th | pvt company | A+S | - | - | - | - | 18 | 13 | 39 | - | - | NR |
| 43 | Nagabushan | 46 | male | M | 10th | real estate | A+S | - | - | - | - | 17 | 38 | 49 | - | - | NR |
| 44 | Shankar | 50 | male | M | 6th | auto driver | - | positive | - | - | - | 18 | 20 | 41 | - | - | NR |
| 45 | Nataraj | 40 | male | M | 6th | manual labourer | A+S | - | - | - | - | 28 | 18 | 44 | - | - | NR |
| 46 | Vetriselvan | 43 | male | M | 10th | truck driver | S | negative | - | - | - | 16 | 20 | 63 | - | - | NR |
| 47 | Krishnan | 44 | male | M | 12th | artist | S | - | - | - | - | 19 | 20 | 26 | - | - | NR |
| 48 | Veeramani | 26 | male | UM | - | manual labourer | S | - | - | - | - | 25 | 26 | 30 | - | - | NR |
| 49 | Mohammed | 28 | male | M | 10th | pvt company | A+S | negative | - | - | - | 11 | 14 | 26 | - | - | NR |
| 50 | Rupkumar | 46 | male | M | 4th | gold smith | A+S | - | positive | - | - | 16 | 20 | 63 | - | - | NR |

| SL.NO | NAME | AGE | SEX | MARITAL STATUS | EDUCATION | OCCUPATION | HRG | PARTNER SCREENING | HIV+HBV POSITIVE | HIV+HCV POSITIVE | HIV+VDRL POSITIVE | LIVER ENZYME LEVELS | | | STI SYMPTOMS | CUTANEOUS FINDING | VDRL |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | | | | | SGOT | SGPT | ALP | | | |
| 51 | Marimuthu | 28 | male | UM | 8th | manual labourer | A+S | - | - | - | - | 18 | 20 | 39 | - | - | NR |
| 52 | Prabhakar | 26 | male | UM | 10th | pvt company | S | - | - | - | - | 14 | 19 | 48 | - | - | NR |
| 53 | Irudhayaraj | 42 | male | M | 7th | manual labourer | A | - | positive | positive | - | 28 | 18 | 94 | - | - | NR |
| 54 | Vijay | 47 | male | UM | - | manual labourer | A+S | - | - | - | - | 19 | 22 | 74 | - | - | NR |
| 55 | Rajan | 35 | male | UM | - | manual labourer | A+S | expired | - | - | - | 12 | 16 | 51 | - | - | NR |
| 56 | Chinivar | 35 | male | M | 5th | manual labourer | A | - | - | - | - | 25 | 28 | 49 | - | - | NR |
| 57 | Ravi | 55 | male | M | - | mason | A+S | expired | positive | - | - | 17 | 13 | 43 | - | - | NR |
| 58 | Ponraj | 27 | male | UM | 12th | pvt company | A | - | - | - | - | 31 | 40 | 48 | - | - | NR |
| 59 | Babu | 35 | male | M | 12th | clerk | A | negative | - | - | - | 23 | 41 | 46 | - | - | NR |
| 60 | Rayar | 39 | male | UM | 9th | manual labourer | A+S | - | - | - | - | 14 | 23 | 41 | - | - | NR |
| 61 | Ram | 37 | male | UM | - | manual labourer | A+S | - | - | - | - | 18 | 40 | 58 | - | - | NR |
| 62 | Ettaiyan | 80 | male | M | 7th | manual labourer | A | expired | - | - | - | 28 | 14 | 40 | - | - | NR |
| 63 | Soundhar | 24 | male | UM | 5th | manual labourer | A | - | - | - | - | 16 | 19 | 31 | - | - | NR |
| 64 | Babu | 41 | male | M | 12th | pvt company | A | - | - | - | - | 13 | 18 | 55 | - | - | NR |
| 65 | Abilash | 39 | male | M | 7th | hotel work | A+S+B | - | - | - | - | 20 | 27 | 38 | - | - | NR |
| 66 | Siva | 43 | male | M | - | manual labourer | A+S | - | - | - | - | 25 | 13 | 46 | - | - | NR |
| 67 | Munusamy | 64 | male | M | 10th | driver | A+S+B | - | - | - | - | 14 | 20 | 62 | NSU+ phimosis | | NR |
| 68 | Somesh | 33 | male | UM | - | painter | A+MSM | - | - | - | - | 18 | 20 | 39 | - | - | NR |
| 69 | Vairavan | 32 | male | UM | 7th | manual labourer | MSM | - | - | - | positive | 17 | 39 | 46 | GUD | - | R |
| 70 | Jeeva | 30 | male | UM | - | manual labourer | - | - | - | - | positive | 16 | 14 | 40 | - | - | R |
| 71 | Malliga | 48 | female | M | 8th | house wife | - | - | positive | - | - | 21 | 33 | 63 | - | - | NR |
| 72 | Tamilarasi | 41 | female | M | - | house wife | - | - | - | - | - | 16 | 20 | 65 | - | - | NR |
| 73 | Sundari | 45 | female | M | 4th | house wife | - | expired | - | - | - | 18 | 24 | 40 | - | - | NR |
| 74 | Shantha | 52 | female | M | 2nd | house wife | - | negative | - | - | - | 25 | 26 | 30 | - | - | NR |
| 75 | Selvi | 47 | female | M | - | manual labourer | - | expired | - | - | - | 16 | 30 | 64 | - | - | NR |
| 76 | Monica | 20 | female | UM | 12th | student | - | - | - | - | - | 11 | 13 | 38 | - | - | NR |
| 77 | Nageshwari | 39 | female | M | 10th | student | - | - | - | - | - | 19 | 14 | 48 | VVC | - | NR |
| 78 | Venktama | 24 | female | UM | - | student | - | - | - | - | - | 25 | 28 | 49 | - | - | NR |

| SL.NO | NAME | AGE | SEX | MARITAL STATUS | EDUCATION | OCCUPATION | HRG | PARTNER SCREENING | HIV+HBV POSITIVE | HIV+HCV POSITIVE | HIV+VDRL POSITIVE | LIVER ENZYME LEVELS | | | STI SYMPTOMS | CUTANEOUS FINDING | VDRL |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | | | | | SGOT | SGPT | ALP | | | |
| 79 | Banu | 29 | female | M | - | house wife | CSW | - | - | - | - | 14 | 12 | 51 | - | - | NR |
| 80 | Palaniammal | 60 | female | M | - | manual labourer | - | - | - | - | - | 28 | 39 | 64 | - | - | NR |
| 81 | Suguna | 35 | female | M | 7th | house wife | - | positive | - | - | - | 13 | 17 | 43 | - | - | NR |
| 82 | Mariyamma | 37 | female | M | - | house wife | - | - | - | - | - | 26 | 33 | 63 | - | - | NR |
| 83 | Geetha | 28 | female | M | - | house wife | - | - | - | - | - | 24 | 16 | 40 | - | - | NR |
| 84 | Alamelu | 30 | female | M | - | house wife | - | - | - | - | - | 28 | 38 | 44 | - | - | NR |
| 85 | Divya | 23 | female | UM | 10th | student | - | - | - | - | - | 31 | 40 | 60 | BV | - | NR |
| 86 | Najima | 35 | female | M | - | house wife | - | - | - | - | - | 16 | 20 | 42 | - | - | NR |
| 87 | Girija | 25 | female | M | - | manual labourer | - | - | - | - | - | 19 | 20 | 46 | - | - | NR |
| 88 | Jaya | 52 | female | M | 9th | house wife | - | - | - | - | - | 21 | 24 | 80 | - | - | NR |
| 89 | Kala | 34 | female | M | 8th | house wife | - | - | - | - | - | 19 | 14 | 41 | - | - | NR |
| 90 | Raji | 20 | female | UM | 10th | student | - | - | positive | - | - | 28 | 19 | 40 | - | - | NR |
| 91 | Lakshmi | 55 | female | M | - | house wife | - | expired | positive | - | - | 19 | 22 | 24 | - | - | NR |
| 92 | Gomathy | 25 | female | M | - | manual labourer | - | - | - | - | - | 28 | 19 | 40 | VVC | - | NR |
| 93 | Rajkumari | 32 | female | M | 6th | manual labourer | - | - | - | - | - | 19 | 22 | 24 | BV | - | NR |
| 94 | Jothi | 35 | female | M | 5th | house wife | - | positive | - | - | - | 16 | 20 | 39 | VVC | - | NR |
| 95 | Valli | 32 | female | M | 8th | house wife | - | - | - | - | - | 30 | 34 | 48 | VVC | - | NR |
| 96 | Lakshmi | 37 | female | M | - | manual labourer | - | - | - | - | - | 18 | 13 | 39 | - | - | NR |
| 97 | Lalitha | 27 | female | M | - | manual labourer | - | - | - | - | - | 17 | 29 | 46 | BV | - | NR |
| 98 | Ponnu | 47 | female | M | - | house wife | - | - | - | - | - | 18 | 20 | 41 | - | - | NR |
| 99 | Sathya | 33 | female | UM | - | manual labourer | - | - | - | - | - | 11 | 17 | 36 | - | - | NR |
| 100 | Bakiyam | 35 | female | UM | - | manual labourer | - | - | - | - | - | 25 | 28 | 48 | - | - | NR |

# KEY FOR MASTER CHART

| | | | |
|---|---|---|---|
| 1. | SL.NO | - | Serial number |
| 2. | HRG | - | High Risk Group |
| 3. | HIV+HBV positive | - | Human Immunodeficiency Virus+ Hepatitis B Virus positive patients |
| 4. | HIV+HCV positive | - | Human Immunodeficiency Virus+ Hepatitis C Virus positive patients |
| 5. | HIV+VDRL positive | - | Human Immunodeficiency Virus+ Venereal Disease Research Laboratory test positive patients |
| 6. | SGOT | - | Serum glutamic oxaloacetic acid |
| 7. | SGPT | - | Serum glutamic pyruvate transaminase |
| 8. | ALP | - | Alkaline Phosphatase |
| 9. | UM | - | Unmarried |
| 10. | M | - | Married |
| 11. | Pvt company | - | Private Company |
| 12. | A+S | - | Alcholic + smoker |
| 13. | A+S+B | - | Alcholic + smoker + blood transfusion |
| 14. | MBT | - | Multiple Blood Transfusion |
| 15. | R | - | Reactive |
| 16. | NR | - | Non-Reactive |
| 17. | BL Leprosy | - | Borderline Lepromatous Leprosy |
| 18. | HZ | - | Herpes Zoster |

| | | | |
|---|---|---|---|
| 19. | T.cruris | - | Tinea Cruris |
| 20. | SD | - | Seborhic Dermatitis |
| 21. | NSU | - | Non Specific Urethritis |
| 22. | GUD | - | Genital Ulcer Disease |
| 23. | CSW | - | Commercial Sex Workers |
| 24. | BV | - | Bacterial Vaginosis |
| 25. | VVC | - | Vulvo Vaginal Candidiasis |
| 26. | MC | - | Molluscum Contagiosum |
| 27. | L scrotum | - | Left Scrotum |

# PROFORMA

**Name:**

**Age/ Sex:**

**Occupation:**

**Address:**

OP no/ Patient ID no:

**Complaints:**

H/o present illness:

H/o vaginal / urethral discharge:

H/o abdominal pain:

H/o dyspareunia:

Menstrual history:

Marital History: Single/ married/ divorced/ widow/ widower

Living together or alone:

Sexual history:

Last marital contact:

Premarital contact:

Extra marital contact:

Previous STI infections / treatments:

**Obstetric history:**

**Past History:**

Tuberculosis:

Diabetes:

Hypertension:

Bronchial asthma:

Previous surgeries:

Blood transfusions:

Jaundice:

**Family History:**

**Personal History:**

IV drug abuse/Smoker/alcoholic

Aberrant sexual practice

**General examination:**

Built:

Pallor:

Jaundice:

Pedal edema:

Generalised lymphadenopathy:

Pulse:

BP:

**Systemic examination:**

CVS:

RS:

Abdomen:

CNS:

**Local examination:**

Female:

Any significant inguinal lymphadenopathy:

Inspection:

Vaginal discharge:

Any genital abnormalities:

Per vaginal examination : Position of cervix and uterus

Cervical motion tenderness

Per speculum examination : Cervical discharge

Cervical erosion

Skin:

Mucosa:

Bones and Joints:

**Male:**

Inguinal lymphadenopathy:

Circumcised/ uncircumcised:

Phimosis:

Urethral discharge:

Subprepucial discharge:

Glans penis:

Testis/ scrotum:

Any ulcer/ erosion/ scars:

Skin:

Mucosa:

Bones and Joints:

**Investigations**

Urine routine:

Urethral/ vaginal / cervical discharge : Grams stain/ wet mount with normal saline

and KOH

Ulcers/erosions: Tzanck smear/ Dark field microscopy/ Grams stain

Urine/ pharyngeal swab/ cervical culture for gonococci

Rapid assay test for HIV 1 and 2

VDRL test for syphilis

HBsAg for Hepatitis B

Anti-HCV antibody for Hepatitis C

Liver Function Tests to study the enzymes levels

Diagnosis:

Clinical:

Microbiologic

# INFORMATION SHEET

- Your specimen (Blood) has been accepted.
- We are conducting a study on HIV detection among patients attending Rajiv Gandhi Government General Hospital, Chennai and for that your specimen (blood) may be valuable to us.
- The purpose of this study is to diagnose early cases of HIV infection with the help of certain special tests.
- We are selecting certain cases and if your specimen is found eligible, we may be using your specimen to perform extra tests and special studies which in any way do not affect your final report or management.
- The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.
- Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.
- The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of investigator Signature of participant

Date:

# PATIENT CONSENT FORM

**Title of the study : *"SEROPREVALENCE OF HEPATITIS B, HEPATITIS C & SYPHILIS AND LIVER ENZYMES LEVELS IN HIV POSITIVE PATIENTS ATTENDING STD CLINIC"***

Name of the participant :

Name of the principal investigator: Dr. Sunitha .N

Name of the Institution : Institute of Venereology,
Madras Medical College &
Rajiv Gandhi Government General Hospital,
Chennai – 3.

***Documentation of the informed consent:***

I ---------- have read the information in this form (or it has been read to me). I was free to ask any questions and they have been answered. I am over 18 years of age and exercising my free power of choice, hereby consent to be included as a participant in the study.

1. I have read and understood this consent form and the information provided to me

2. I have had the consent document explained to me

3. I have been explained about the nature of the study

4. My rights and responsibilities have been explained to me by the investigator

5. I agree to co operate with the investigator and I will inform him/her immediately if I suffer unusual symptoms

6. I have not participated in any research study at any time

7. I am unaware of the fact that I can opt out of the study at any time without having to give any reason and this will not affect my future treatment in this hospital

8. I hereby give permission to the investigator to release the information obtained from me as a result of participation in this study to the sponsors, regulatory authorities, Government agencies and institutional ethics committee. I understand that they are publicly presented.

9. My identity will be kept confidential if my data are publicly presented

10. I am aware that if I have any question during the study, I should contact at one of the addresses listed above. By signing this consent form I attest that the information given in this document has been clearly explained to me and apparently understood by me, I will b given a copy of this consent document.

**Participant initials:**

**For adult participants:**

Name and signature/ thumb impression of the participant (or legal representative if participant incompetent)

| _____ | _____ | _____ |
|---|---|---|
| Name | Signature | Date |

Name and signature of impartial witness (required for illiterate patients):

| _____ | _____ | _____ |
|---|---|---|
| Name | Signature | Date |

Address and contact number of the impartial witnesss :

Name and signature of the investigator or his representative obtaining consent:

| _____ | _____ | _____ |
|---|---|---|
| Name | Signature | Date |

# ஆராய்ச்சி தகவல் தாள்

**தங்களது ரத்தம் இங்கு பெற்றுக் கொள்ளப்பட்டது.**

சென்னை அரசு பொது மருத்துவமனையில் பல்வினைத்துறைக்கு வரும் எச்.ஐ.வி. பாஸிட்டிவ் புற நோயாளிகளிடம் ஹெபடைடிஸ்-பி, ஹெபடைடிஸ்-ஸி, ஸிஃபிலிஸ் மற்றும் கல்லீரல் சம்மந்தப்பட்ட மாற்றங்கள் பற்றிய நோய் பரவியிருக்கை குறித்த ஆராய்ச்சி.

நீங்களும் இந்த ஆராய்ச்சியில் பங்கேற்க நாங்கள் விரும்புகிறோம். இந்த ஆராய்ச்சியில் உங்களுடைய இரத்தத்தை சில சிறப்புப் பரிசோதனைக்கு உட்படுத்தி அதன் தகவல்களை ஆராய்வோம். அதனால் தங்களது நோயின் ஆய்வறிக்கையோ அல்லது சிகிச்சையோ பாதிப்புக்கு ஏற்படாது என்பதையும் தெரிவித்துக் கொள்கிறோம்.

முடிவுகளை அல்லது கருத்துக்களை வெளியிடும் போதோ அல்லது ஆராய்ச்சியின் போதோ தங்களது பெயரையோ அல்லது அடையாளங்களையோ வெளியிடமாட்டோம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

இந்த ஆராய்ச்சியில் பங்கேற்பது தங்களுடைய விருப்பத்தின் பேரில் தான் இருக்கிறது. மேலும் நீங்கள் எந்த நேரமும் இந்த ஆராய்ச்சியலிருந்து பின் வாங்கலாம் என்தையும் தெரிவித்துக் கொள்கிறோம்.

இந்த சிறப்புப் பரிசோதனைகளின் முடிவுகளை ஆராய்ச்சியின் போது அல்லது ஆராய்ச்சியின் முடிவின் போது தங்களுக்கு அறிவிப்போம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

**ஆராய்ச்சியாளர் கையொப்பம்** **பங்கேற்பாளர் கையொப்பம்**

**தேதி :**

# ஒப்புதல் படிவம்

ஆராய்ச்சி தலைப்பு : சென்னை அரசு பொது மருத்துவமனையில் பால் வினை துறைக்கு வரும் எச்.ஐ.வி. பாஸிட்டிவ் புற நோயாளிகளிடம் ஹெபடைடிஸ்-பி, ஹெபடைடிஸ்-ஸி, ஸிஃபிலிஸ் மற்றும் கல்லீரல் சம்மந்தப்பட்ட மாற்றங்கள் பற்றிய நோய் பரவியிருக்கை குறித்த ஆராய்ச்சி

பெயர் : தேதி :

வயது : புறநோயாளியின் எண் :

பால் : ஆராய்ச்சி சேர்க்கை எண் :

இந்த ஆராய்ச்சியின் விவரங்களும் அதன் நோக்கங்களும் முழுமையாக எனக்கு தெளிவாக விளக்கப்பட்டது.

எனக்கு விளக்கப்பட்ட விஷயங்களை நான் புரிந்து கொண்டு எனது சம்மதத்தை தெரிவிக்கிறேன்.

எனக்கு இரதத்தத்தில் பரிசோதனை செய்து கொள்ள சம்மதம்.

இந்த ஆராய்ச்சியில் பிறரின் நிர்பந்தமின்றி என் சொந்த விருப்பத்தின் பேரில் தான் பங்கு பெறுகிறேன் மற்றம் நான் இந்த ஆராய்ச்சியிலிருந்து எந்நேரமும் பின்வாங்கலாம் என்பதையும் அதனால் எந்த பாதிப்பும் ஏற்படாது என்பதையும் புரிந்து கொண்டேன்.

நான் எச்.ஐ.வி. கிருமி தாக்குதல் ஹெபடைடிஸ்-பி, ஹெபடைடிஸ்-ஸி, ஸிஃபிலிஸ் மற்றும் கல்லீரல் சம்மந்தப்பட்ட மாற்றங்கள் குறித்த இந்த ஆராய்ச்சியின் விவரங்களை கொண்ட தகவல் தாளைப் பெற்றுக் கொண்டேன்.

நான் என்னுடைய சுய நினைவுடன் மற்றும் முழு சுதந்திரத்துடன் இந்த மருத்துவ ஆராய்ச்சியில் என்னை சேர்த்துக் கொள்ள சம்மதிக்கிறேன்.

ஊசி மூலம் இரத்தத்தை பரிசோதனைக்கு எடுத்துக் கொள்ள சம்மதிக்கிறேன். அப்போது ஏதேனும் பின்விளைவுகள் (மயக்கம், தலைசுற்று) ஏற்படலாம் என மருத்துவர் மூலம் புரிந்து கொண்டேன்.

**கையொப்பம்**

**INSTITUTE ETHICS COMMITTEE**
**MADRAS MEDICAL COLLEGE, CHENNAI-3**

EC Reg. No.ECR/270/Inst./TN/2013
Telephone No.044 25305301
Fax: 044 25363970

**CERTIFICATE OF APPROVAL**

To

Dr.Sunitha.N.
Post Graduate M.D.(Dermatology)
Madras Medical College, Chennai -3.

Dear Dr. Sunitha.N.

Seroprevalence of Hepatitis –B Hepatitis C and Syphilis & liver enzyme levels in HIV positive patients attending STD clinic at Madras Medical College, Chennai " No.09092014.

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|---|---|
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| 2. Dr.V.Vimala,M.D. Dean,MMC,Chennai-3 | : Deputy Chairperson |
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| 5. Prof. Raghumani, Prof/HOD. Surgery | : Member |
| 6. Prof. Md.Ali. Prof. and HOD of MGE,MMC | : Member |
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| 12.Thiru S.Govindasamy,BA.BL. | : Lawyer |
| 13.Tmt.Arnold Sauline,M.A.MSW | : Social Scientist |

We approve the proposal to be conducted in its presented in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any6 changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

Member Secretary, Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
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CHENNAI-600 003